The Compensation Neurosis*

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There is no more vexing problem in all of forensic medicine than the problem of the Compensation Neurosis. Dorland's Medical Dictionary describes this condition as "the psychoneurotic phenomena developing after an accident in persons who are insured, or who believe that by being ill they may secure any compensation." Another definition might read, "a complex interpersonal situation in which the possibility of reward through litigation is important in the perpetuation or exaggeration of physical or psychiatric symptoms." From the standpoint of the psychiatrist called upon to help restore lost function or facilitate rehabilitation in general, the individual with Compensation Neurosis constitutes a most difficult patient. For the attorney, such patients constitute a complex legal, ethical and moral dilemma.

This paper will consider three case presentations concerning patients studied extensively in a general hospital. The discharge diagnosis in each instance was Compensation Neurosis. The intricate relationship between injury, psychological predisposition of the patient, and interaction with an attorney as a compounding factor will be discussed.

Case Study Number I

Mrs. R., a forty-four-year-old married and separated mother of eight children, was transferred from the Orthopedic Department to the Psychiatric Department of the general hospital after a two-week period of study. During the time of the patient's hospitalization on the orthopedic service, detailed physical and laboratory examinations failed to reveal

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any organic basis for the patient's many complaints. Her physicians felt that the patient was either malingering (that she was pretending to have symptoms which she really did not have) or that she was suffering from some kind of Compensation Neurosis. Patient's chief complaints were: "I hurt all over. Every bone in my body aches. I've been getting steadily worse for seven months. Since I slipped on the ice, I've been going down hill." Physical, neurological and physical + neurological examinations were all negative. The patient was a person who felt that she was so sick as to be unable to leave her bed. There were, however, no objective evidences of any serious physical difficulty. X-ray examinations revealed some early arthritis, but this arthritis was felt to be in no sense of sufficient magnitude to explain her difficulties The patient left her bed only to go for meals and to the toilet. She complained continuously of pain. She felt very bitter about the inability of the physicians in the hospital to find the cause of her difficulty.

The patient's past history was quite interesting. She was the oldest of five children born to a farm family. She was compelled to quit school in the fifth grade because she was needed at home to help with the care of the smaller children. She experienced a good deal of bitterness to have been singled out to assume many responsibilities not shared by the younger siblings. At age nineteen, she became pregnant and was compelled to marry a local boy who was also nineteen. The marriage was a source of considerable humiliation to her. One year later, a second child was born, and in the succeeding ten years, six more children were born, making a total of eight. The patient's life was very difficult and her husband's income was marginal and unpredictable. There were frequent family battles because of the husband's drinking, irregular work habits, and occasional staying out all night. Patient was deserted twice by her husband, the first time two years before her accident. On this occasion, the husband was arrested for non-support and returned to the family. Fifteen months later, approximately six months prior to her accident, the husband deserted the family again, this time failing to return. The patient was compelled to seek some restaurant work, she also

received some aid for dependent children. This was the nature of things in her life at the time of the accident.

Details of the accident seemed rather simple. On a rather snowy day the patient lost her footing in the entranceway of a department store. She did not lose consciousness, but she apparently twisted her ankle. She went first to the First Aid station in the department store where a nurse examined her foot and reassured her that nothing was wrong. The patient left the store somewhat indignantly. Then, when her foot swelled up to some degree later in the afternoon, the patient went immediately to an attorney. The attorney advised that she receive a complete set of X-rays; the patient was referred to a local physician who obtained X-rays of her spine, extremities, and skull, as well as of the ankle involved. These X-rays revealed some arthritic condition which had existed prior to the accident. During the next several weeks the patient was involved in several more physical examinations and an attempt was made by the department store attorneys to settle the claim. Regarding delay in settlement, the patient commented: "My lawyer told me we had better wait and see what developed. He told me about cases in which things came many months later." In the succeeding months, patient developed a myriad of somatic complaints and she was confined to her bed. She remained more or less bedfast during the succeeding six months, during which time her mother came to live with her and assumed responsibility for her children. The litigation remained unsettled at the time of her hospitalization in the general hospital.

It is possible to achieve an understanding of this patient's problem on many levels. For one thing, she clearly wanted and needed money. But this was not the whole story. She was a person to whom life had not been especially kind, and she had a long-sustained resentment of her overall life situation. Her unhappy childhood, her disappointing marriage, and the final desertion by her husband left her with a deep and sustained feeling that "the world certainly owes me something." The casual treatment by the nurse in the department store seemed a vignette of the casual treatment which she had received all of her life. The animosity which she developed

towards the department store was in part a summary of the animosity which she had been building up for many years. Her life situation directed her in such a way as to be litigiously inclined at the time of her accident. Still another level of understanding is possible in terms of the living arrangement which had been created by the virtue of the patient's illness. In a way, history reversed itself. Some thirty years earlier, the patient had had to give up her schooling in order to go out and help the mother. In the present situation she was helpless and her mother had to care for both her and for her children.

Psychiatrists confronted by a persisting symptom in their patient will often ask; what would my patient's life be like without this symptom? What would recovery mean to this lady? She would need to return to work. She would need to give up her relatively dependent role with regard to her mother. She would need to acknowledge that really, nobody owed her very much. In this instance, the likelihood that this patient would pick up the pieces of her life seems slight. She certainly has no chance to do so until her legal problems are settled.

Case Study Number II

The patient, a fifty-five year old housewife in a small town, was injured in a bus accident. By objective standards, her injuries were slight, but in the five months following this injury she developed a steady progression of symptoms which led ultimately to almost complete incapacity. While riding on the bus, there was an abrupt stop and she fell to the floor of the bus. She did not lose consciousness, but did sustain a large bruise on her thigh which healed very slowly and some discoloration was present one month after her fall. Patient subsequently began to develop what she felt was weakness in her leg, and she began to stumble and fall during the weeks after the accident. The family doctor took X-rays of the patient's legs and spine but was unable to demonstrate any evidence of orthopedic pathology. She was then referred to a neurologist for consideration of the possibility of a ruptured intervertebral disc. He reported, "There is no evidence of organic pathology. Signs indicate a great deal of emo-

tional overlay in this patient's difficulties." Patient's disturbed gait persisted and reached a degree where it was almost impossible to walk. Three months after her fall she was admitted to the Psychiatric Service for study. At that time, she could bend her knees only slightly and she claimed to be unable to turn her foot so as to walk on the sole. Patient was studied quite intensively. She was given a series of sodium amytal interviews. While under the influence of this medication it was possible for her not only to straighten her knee and ankle, but also to walk normally. When the drug wore off, however, her foot would turn inward and she was grotesquely crippled. Patient's attitude towards her infirmity was one of rather calm resignation. She took her infirmity very placidly.

The patient's past history was quite interesting and it was learned that a recently divorced stepdaughter with several children had come to live with the patient and her husband. Patient had been more or less compelled to resume the duties of mother for her stepdaughter's children, three, all under the age of seven. She resented the new task, but was unable to do much about it since her husband gave her no support in this issue. Patient's illness solved this problem. By being injured and the victim of an accident, she could not be blamed for her inability to assume the task that she did not want. Other aspects of the patient's personality and history seemed to make her especially vulnerable for this sort of difficulty. In general throughout life she had been a very unhappy person. She had married for the first time at forty, after having kept house for her aged parents. Marriage to her husband had provided the first period in her life when she felt in any way justly treated. The return of her stepdaughter, her husband's child by his first wife, threatened, she felt, her role in her husband's home. The patient was a retiring person who had little heart for open battles and it was not possible for her to openly oppose the return of the stepdaughter. Furthermore, ethics seemed to obligate the task. Somehow or other, the problem was resolved by an accident which made her the dependent one. She felt, partly unconsciously, that the legitimacy of her act would somehow or other be attested to if there were sufficient legal damage reward.

Case Study Number III

Patient is a thirty-five year old factory worker, married and the father of three children. Hospitalization was precipitated preparatory to a hearing in a damage suit. Patient claimed that he had sustained almost complete blindness following an injury to his eye resulting from work injury. Diagnosis of hysterical blindness had been made by two ophthalmologists. The patient described a visual loss which might thus be described as allowing only a tubular kind of vision. The blindness described was the same in both eyes. The patient was unable to see anything that was not directly in front of him. Objects to the right and left could be seen only if the patient turned his head in that direction. The patient and his attorney sought damages totaling approximately forty thousand dollars.

There was no question but that the patient had sustained some injury at work. A small glass fragment from an exploding bulb struck the patient's right eye. He was hospitalized at company expense. The eye was covered for several days with a patch. When the patch was removed, however, the patient reported that his vision was quite blurred, subsequently that he could not see except in a very narrow visual range, and subsequently still that the difficulty had spread to the other eye.

The patient's history revealed an almost life-long tendency toward illness and incapacity. He had found it necessary to leave school in the tenth grade because his classes made him nervous. He enlisted in the Army Air Force but after one year was released from the service with a general discharge. He spent several years wandering about the country doing odd jobs. During the seven years between the time of his discharge from the Army and his marriage, he held a large number of jobs. He rarely worked longer than six months on any job. His usual reason for leaving was either nervousness or drinking on the job or some argument with an employer. He married a woman four years older than himself and they quickly had three children. During the eight years of their marriage, the patient's wife had worked most of the time. He was a generally unsatisfactory provider and he was frequently ill with a wide variety of complaints. He had worked

for approximately four months in the job which he held at the time of the injury to the eye.

This patient was an individual with a minimal capacity to deal with stress. He frequently left places of employ because he felt they constituted some danger to his general health. He had many fundamental doubts about the health and vitality of his own body and it took little to convince him that he was incapacitated. He was a person vulnerable to an intense type of reaction to any injury. Encouragement by his attorney to consider the possibility of permanent blindness fell upon most receptive ears.

The attorney's obligation to "do the best possible" for his client is ordinarily construed to mean that the attorney's obligation is to obtain as much money as possible for his client. However, incapacity associated with Compensation Neurosis can last a lifetime and can result in loss which considerably overshadows the gain with litigation. Some patients never recover from the complaints which become magnified in the face of possible compensation. What is the lawyer faced with the compensation problem to do? The development of a neurosis which lasts a lifetime and incapacitates an individual offsets whatever possibilities a compensation may provide. Nevertheless, pensions, compensations, and litigations for damages, seem absolute as requirements in a just society.

Medical management of the patient suffering from a Compensation Neurosis may be pertinent. It has been the observation of the author and many other psychiatrists that it is almost impossible to offer meaningful psychotherapy to a patient suffering from a Compensation Neurosis prior to settlement of litigation. For this reason, when a Compensation Neurosis is definitely diagnosed, prompt settlement of all litigation, one way or another, is usually recommended. The longer that settlement is delayed, more likely the psychologically predisposed individual will fail to recover completely from his emotional illness. Secondly, patient is advised to return as rapidly as possible to work or to some other kind of gainful activity. Rehabilitation following injury should begin quickly. The long-immobilized individual, like the freight train which is at a dead stop, has much inertia to overcome.

Summary

This paper has reviewed some aspects of a very complex situation which is labeled "Compensation Neurosis." All too often, the Compensation Neurosis with its sometimes devastating toll of life-long incapacity, is the result of the unique interaction and subtle encouragement of the well-intentioned attorney upon a psychologically predisposed individual. The psychological meaning to an individual of an injury, and the long-term as well as the short-term aspects of its settlement, are facts which the lawyer must weigh. The importance of avoiding undue delay in settlement of damages for personal injury and the importance of early mobilization of the patient in the direction of useful activity has been stressed.

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Determination of the Time of Death*

A Consideration of Post-Mortem Physical Changes

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An average of a million and a half persons die each year in the United States. Of that number, approximately 300,000 fall into the category of coroner's or medical examiner's cases. These deaths must be investigated to ascertain whether death was the result of suicide, accident, homicide or natural causes. The deceased is originally viewed or cursorily examined by a lay person, a friend, relative, police officer or medical investigator. Subsequently, the body may be observed, examined or subjected to a necropsy by either a physician or the pathologist. The vast majority of cases, however, are never seen or examined by the forensic pathologist or other trained physician whose examination could be of value to estimate the time when death had occurred.

This gives rise to several questions. Can the police officer, medical investigator, or other lay observer recognize and record facts pertaining to the dead body that will be of value in determining the time of death, employing only a visual or manual inspection of the body? Can the observations made by these lay persons be of significance and value to the medical expert who at a later time may be called upon to interpret

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the observations and determine how long the person has been dead? The following dissertation is an attempt to outline the physical changes that take place in the human body after death and to indicate their value and significance.

Most of the forensic investigators who have written about the subject have based an estimation of the time elapsed since death upon the following physical alterations: appearance of livor mortis, rigor mortis, changes in the eyes and putrefaction. Complete agreement is lacking as to the times of appearance of these external signs.

The opinion of a physician who reports an estimation of the time of death is frequently reported by the newspapers. Such reports often misquote the opinion that has been given. Unwary persons who do not realize the magnitude of the variations in the actual time or order of the appearance of the external signs or who are subjected to pressure from interested officials may make unqualified and unsupportable statements fixing an exact time of death. These unfortunate statements may mislead investigators, attorneys or public officials and may create a false impression in the eyes of the public as to the true capabilities of scientists in the field.

In order to properly present a discussion regarding the scientific manner of determining the time of death, an extensive review of the literature was made. It was found that most of the publications appeared in foreign journals. A comprehensive survey of the work accomplished could not be located in the English literature.

Instead of presenting an appraisal of the published investigations at this time, the observations detailed in this discussion represent the current data obtained from persons whose deaths were investigated as a matter of routine in the office of the Coroner of Hamilton County, Ohio.

The evaluation of changes in the body requires a careful consideration of the variables normally encountered in the investigation of deaths from any cause. One must record the pertinent variables such as the ambient temperature, the relative humidity, the covering or clothing of the body, the general structure as well as the supporting structure of the body, the presence and activity of scavengers which consume flesh, the wind currents and other such influencing factors. One

must also consider additional items such as the events which occurred prior to death: the existence of injury, the relationship of injury and death, the interval between injury and death, the cause, mode and manner of death, the musculature activity of the deceased prior to injury or death and the health of the person before death.

To derive the maximum value from observations such as post-mortem rigidity and changes in the appearance of the eyes, it is essential that the deceased be examined at the site of death and before the body has been subjected to any physical movement. In this manner, the position of the eyelids, the degree of drying and the extent and distribution of rigor mortis may be determined. In the experience of the authors, it is impossible to evaluate these changes properly if the body is subjected to manipulation during removal from the scene of death, during transference to the morgue or during the removal of the clothing from the body in the morgue.

The Eye

The film that appears over the external surface of the eyeball has been observed to appear in as short a time as 10 minutes after death in a person who collapsed and died in the presence of an observer (HPL). The eyelids were open. The weather was cold and dry. Dilatation or contraction of the pupils of the eyes after death is related only to the initial flaccidity and later rigidity of the muscle of the iris. Discoloration of the sclera resulting from drying of the tissue appears over a period of several hours and is dependent upon the position of the eyelids. Cloudiness of the cornea appears in 12 to 24 hours and is related to the position of the eyelids, the temperature, humidity and air currents. Its appearance has been noted in 1 hour and 45 minutes when the eyelids were open. Complete opacity of the cornea appears by the third day after death. Bulging of the eyeballs (exophthalmus) and retraction or sinking of the eyeballs into the sockets (enophthalmos) result from putrefaction and mummification as the deterioration of the body progresses. Ophthalmoscopic examination of the retina and the retinal vessels, noting the segmentation of the blood columns and their movement in the retinal veins, is of value only within the

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The Eye

The film that appears over the external surface of the eyeball has been observed to appear in as short a time as 10 minutes after death in a person who collapsed and died in the presence of an observer (HPL). The eyelids were open. The weather was cold and dry. Dilatation or contraction of the pupils of the eyes after death is related only to the initial flaccidity and later rigidity of the muscle of the iris. Discoloration of the sclera resulting from drying of the tissue appears over a period of several hours and is dependent upon the position of the eyelids. Cloudiness of the cornea appears in 12 to 24 hours and is related to the position of the eyelids, the temperature, humidity and air currents. Its appearance has been noted in 1 hour and 45 minutes when the eyelids were open. Complete opacity of the cornea appears by the third day after death. Bulging of the eyeballs (exophthalmus) and retraction or sinking of the eyeballs into the sockets (enophthalmos) result from putrefaction and mummification as the deterioration of the body progresses. Ophthalmoscopic examination of the retina and the retinal vessels, noting the segmentation of the blood columns and their movement in the retinal veins, is of value only within the

first half hour after death. The dark grey appearance of the macula of the eye is reported to indicate an interval of several hours from the time of death.

The changes in the eyes are of value to indicate that death has occurred and that it has taken place within a few minutes of the observation (segmentation and movement of blood columns in the retinal veins), within several hours (clouding of the cornea, color of the macula, decreased intraocular pressure) or within several days (enophthalmos).

The Skin

Pallor of the skin, mucous membranes, and nail beds is the external evidence of cessation of the circulation and is of no assistance in measuring the interval since death.

Livor mortis or post-mortem lividity is the result of settling of the blood into the dependent parts of the body. The fluid blood settles by the action of gravity. Lividity not infrequently may appear in the terminal period in severely ill patients due to a grossly deficient circulation. Lividity may fail to appear after death under the following circumstances: Where there is an absence of blood in deaths resulting from exsanguination and where there is severe anemia and a deficiency of blood and blood pigment. Thus, absence of post-mortem lividity does not signify that death has occurred recently.

The onset of lividity may be influenced by such factors as the manner of death and environmental conditions. Lividity is reported to range from 20 minutes to 4 hours in its appearance after death. Livor reaches its maximum intensity in 8 to 12 hours, it becomes fixed in the dependent portions of the body in 3 to 12 hours and will not change position or disappear upon movement of the body. Lividity may blanch under external pressure for as long as 10 to 12 hours following death after which pressure will have no effect. Lividity appears in those dependent portions of the body not subjected to external pressure from supporting objects or constricting clothing. If the position of the body is changed so that non-livid areas become dependent, livor will appear in these areas in 6 to 12 hours after death. Accurate observations upon the

degree of lividity can be made only in persons having light-colored skin.

In a series of 100 cases, lividity was noted to vary in degree from slight to marked even when fully developed. These observations were made on persons dead 18 hours or longer. Where the initial observation discloses that livor is not yet present and the absence of lividity is to be used as a factor in estimating the time of death, it is essential that the subsequent observations be carried out at intervals of 30 minutes to an hour for a significant period of time. Thus, the observer will be able to define the time of onset, the period necessary to reach maximum intensity and the interval at which livor mortis becomes fixed, no longer responding to external pressure. In this manner an estimation of the time since death occurred will have greater significance.

Drying of the skin and cutis anserina (goose flesh) depend upon external factors that are extremely variable and are of no value in arriving at an estimation of when death occurred.

Rigor mortis

Observations of the presence and distribution of post-mortem rigidity have been considered and reported by many investigators as the most important of the external changes to be used in estimating the time interval since death. As noted previously it is of great value to know whether or not rigor has been disturbed by physical manipulation particularly in those cases where there is unequal distribution or even absence of rigidity.

In a study to be reported subsequently, it was recorded that in the early period after death, the initial rigor upon being broken during movement of the body would return in the disturbed muscles to a considerable degree. The observations were made with particular attention to rigidity involving the joints of the fingers and elbows.

Most authors report that the muscles of the body develop rigidity in a sequential progression from the head downward. Some state the belief that rigor involves the lower extremities prior to the upper extremities. Other observers suggest many variations in the suggested order of development.

In a large series of cases, where there was an absence of manual disturbance to the development of post-mortem rigidity, observations indicated the following order in the involvement or envelopment of the body by rigor, (1) the jaw, (2) the neck and fingers, (3) the wrists, (4) the elbows, (5) the shoulders and knees, and (6) the hips. Rigidity in the muscles of the eyelids, toes, thorax and abdomen was too difficult to detect and evaluate. It appears reasonable to assume that rigor mortis begins to develop simultaneously in all of the muscles of the body within the same definitive period after death has occurred. The variations that seem to exist are due in part to the sparsity of the observations and in part to the degree of attentiveness in noting the sequence of the alterations. Thus, an important factor is the actual mass of the muscle being considered. For example: The mass of muscle in the eyelid not only is small, but is extremely difficult to assess. Thus, rigor may be complete and yet the observer would be unable to detect the rigidity. Also, the mass of muscle in the thigh is so great, that even a physically strong man is unable to break the rigor once it has reached its maximum development. Thereby the unskilled or infrequent observer is more greatly impressed by rigor mortis of the extremities than he is by the rigor involving the little finger of the hand.

Immediately after death, there is a brief interval of time during which all muscles of the human body are completely flaccid. Thereafter, as a result of chemical changes within the musculature of the body, there is the gradual and progressive onset of the change designated as rigor mortis. The time interval of the post-mortem flaccidity varies and is influenced by such factors as the physical activity of the person prior to death, the environmental conditions and the manner of death.

Post-mortem rigidity has been reported to appear within minutes after death in persons who have been engaged in heavy or strenuous physical exertion or exercise. Rigor has been noted to appear in all the areas previously enumerated within periods of time ranging from 85 minutes to 135 minutes in two persons who died of heart disease and cirrhosis of the liver respectively, without any prior muscular activity.

There are insufficient data pertaining to a delay in the development of rigidity and the circumstances relating to the reasons for the delay.

In a series of cases where the time of death was known, the degree of the development of rigor mortis was noted at the beginning and at the termination of the observations. The interval between death and the first observation ranged from 63 to 199 minutes. The final observations were made at intervals ranging from 11 to 31 hours. All cases which showed no rigor initially had complete rigor at the final examination. Thus, complete rigor persisted for as long as 31 hours after death. In several cases where the exact time of death was unknown, but where a probable time of death was presumed, rigor was present for as long as 38 hours.

The order of resolution of rigor mortis is not precisely defined, although many observers report its disappearance is in the same order as its onset. Rigidity was noted to resolve with the onset of early putrefactive changes. From the observations made in the described series of cases, it was not possible to define the duration or interval for complete resolution after full envelopment of the body by rigor. Factors such as temperature and muscular mass undoubtedly are of importance.

Decomposition and Destruction

The terminal process of putrefaction appears in the body under ordinary environmental conditions after a certain period of time, usually after more than 24 hours. Beginning putrefaction, characterized by greenish discoloration of the abdomen, has been observed in 21 and 26 hours after death. Many factors influence the onset of the putrefactive changes such as environmental temperature, humidity, air currents, post-mortem lividity and post-mortem movement of the body. The latter tends to disseminate organisms which hasten the deterioration. Putrefaction often is observed earlier and progresses more rapidly in the areas of marked livor mortis.

Different geographic areas will tend to give differences in the rates of putrefaction. A high atmospheric temperature will cause putrefaction more rapidly than a cool temperature. Bodies submerged in water will exhibit rather advanced pu-

trefaction from the level of the pelvis to and including the head and upper extremities as these areas float in the surface water having a higher temperature. On the other hand, the lower extremities floating in deeper and colder water will remain relatively well-preserved. Such putrefactive changes have been observed in a significant number of cases and are apparent within a period of 30 hours after the body has gone into the water in the summer season. Similar, but less advanced, putrefaction has been noted in the same period of time in the dependent livid areas of bodies found in open places such as parks or fields. It follows, therefore, that putrefaction does not provide a means for determining the time of death except within a very broad range of time, and with environmental conditions as an essential factor to be considered.

Skeletonization of the body usually requires a much longer period of time and is influenced by the environmental temperature and access to the body by flesh-consuming scavengers. A woman, who died in bed, was reduced to a skeleton except for the skin over the anterior portion of the body within 6 weeks. Partial skeletonization has been observed after 6 months of submersion. Complete skeletonization of a body of a young man who committed suicide in a wooded area was noted in a period of 5 months from July to November. During the summer months, there is great activity among ants, flies, beetles, mice and rats and destruction of the body can proceed with unbelievable rapidity. During winter such activity is not present and a body may be found without significant alteration even after several months.

Mummification generally is observed during hot and relatively dry weather and where there are sufficient air currents to facilitate the loss of water. Adipocere formation depends upon the presence of moisture and minerals to react with the fat to form soaps. These processes have such a wide range of variation that they are of little value for determining the time of death except under unusual conditions.

Summary

Many factors play an important role in the time of onset, the degree of development, the duration and the type of

changes that occur in the human body after death. Some of the factors are environmental conditions, general body structure, injury or disease, ante-mortem activity of the deceased and post-mortem handling of the body.

Conclusions

The exact time of death cannot be determined by visual or manual examination of the dead body.

The duration of the time interval after death may be estimated within broad limits by the consideration of the influencing factors in conjunction with the changes in the body.

The fewer the variable factors and the greater the number of body changes evaluated the greater will be the accuracy of the estimate of the time of death.

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Evidential Aspect of Color Photography*

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The Color Photography Revolution

A revolution in color photography is now in progress. The speed of color film has been raised by the manufacturers to such an extent that it is literally possible to take color pictures with the light of a single candle. This phenomenal development in the speed of color film has removed the many early limitations on its use and has placed color film on a par with black and white mediums.

The second major development in color film was the addition of the dye coupler to the film itself, thus removing color processing from the confines of the manufacturer's own laboratories. As a result, at the present time even the amateur photographer, using simple equipment and techniques, is now able to process color film and to make color prints of very satisfactory quality.

Since the advent of color film in 1935, the public, as well as many specialized services, have used color photography extensively. The ability to do home processing and home color printing should result in a tremendous upsurge in the use of color film materials.

Color photography as an investigative technique (1) and as an instrumentality of proof (2) has received a great deal of attention and almost universal acceptance.

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Limiting Factors

At the outset, one should bear in mind that actually there is no available process of color photography which can be said to give entirely accurate and repeatable reproduction of color. Color films give satisfactory color rendering for their intended purpose but the present art does not make it possible to design materials suitable for precise color records or matching or measuring color (3).

There are quite a number of conditions which will affect true color rendition. These color variables, which must be recognized, are generally categoried as follows: (1) Illumination; (2) Exposure; (3) Contrast; (4) Processing; (5) Reflections; (6) Psychological and Visual Factors; and (7) Impurities in, and Deterioration of Dyes. In analyzing a color film or print, each one of these variables must be explored to determine its effect on color rendition.

Factor of Realism

The color content of a film or print becomes significant only when *color* as such has probative value. If a color film or print is introduced to portray an object, person, or scene, and not to identify a particular color value, then general rules pertaining to photographic evidence will be applicable. If such color exhibit is offered to prove a certain color or combination of colors, then obviously it should be necessary to establish the authenticity of color values.

Now we have used black and white photographs for so long that we accept them as the real thing. Actually, black and white photography is considered an abstract medium and does not represent reality as such. Color photography is more realistic than black and white photography because, besides perspective and contrast, it adds another dimension, that of color. Once we forget its glamorous aspects, we shall accept color photography as the closest proximation to reality.

The inherent realism of color photography has been urged, unsuccessfully, as a reason for rejecting black and white photographs of the same subject (4). When color films become the accepted standard of photography, it is conceivable

that the courts will sustain objections to black and white photographs lacking the realism of color pictures.

Meaning of Terms

When we talk of the new color films and prints, we should be careful to use the term "color photography" to distinguish it from the phrase, "colored photographs." Color photography has been defined as the production of color images by physical or chemical techniques other than hand coloring; any mechanical process which records the form and the color of an object, person, or scene by means of photography (5). Colored photographs, on the other hand, in the mind of the public, refer to a hand-painted picture (6).

Prior to the advent of color photography, the courts had been rather liberal in permitting in evidence colored photographs or diagrams, over the objection that such type of exhibit was inflammatory (7). This survey of cases decided prior to the advent of color film and color prints indicates that *color* as such does not frighten the courts.

Early Use in the Judicial Process

The first reported case involving the admissibility of color photographs, decided in 1943, is Green v. City and County of Denver (8), in which the trial court allowed color prints of suspected contaminated meat lying alongside fresh meat to prove that the suspected meat was putrid. The Green case represents an ideal situation calling for the use of color film. The color of the putrid meat was of definite probative value and, therefore, the use of the color film for comparison purposes had a definite cogency.

A few years later, in 1947, a Texas court in a personal injury suit allowed in evidence a "technicolor" motion picture film to show the manner of operation of a machine (9). However, the *color* aspects of the moving picture were not discussed by the court.

These early cases illustrate the early use of color films in the courts. As would naturally be expected, their use in homicide cases became quite pronounced and led to the outcry, "gruesome and inflammatory!"

Admissibility of Color Films

When we consider the admissibility of color films and prints in evidence, we should make certain general observations:

(1) Many times the appellate courts have before them for decision the admissibility of color films but the appellate decision makes no mention of the fact that color film is involved.

(2) The admission of all photographic evidence is subject to the discretion of the court. The reviewing court acts only on the question whether the trial court abused its discretion in the admission or rejection of color films. Consequently, a decision in one case is not necessarily controlling in another.

(3) In most cases, the color content of the film is not material and, therefore, color becomes an incidental fact.

A review of the cases indicates that to date the courts have considered the admissibility of color photography in the following types of cases:

(1) *Homicide.* Color photography has been used to a great extent in homicide cases to depict the scene of the crime, including the location of the body, and to portray the body or parts thereof in order to bring out the nature of the wounds. The courts have sanctioned the use of such evidence for such purposes, over the customary objection that color films are gruesome and inflammatory (10). State v. Bischert (11), is the only case found in which an appellate court was of the opinion that it was prejudicially erroneous to admit in evidence color slides of a small child to illustrate the testimony of a pathologist.

(2) *Other Criminal Cases.* In two rape cases (12) and one felonious assault case (13) color photographs of the victim were held admissible to show body condition, a relevant fact in each case.

(3) *Personal Injury Cases.* The use of color photography in personal injury cases to illustrate the nature of personal injuries was to be expected. In the only two

cases reported to date (14) the courts have shown a liberal tendency to admit such photographs in evidence, over the usual objection that such type of proof is inflammatory per se.

We can predict that the courts will soon be passing on the use of color films in other legal fields.

Appraisal and Prognosis

Color photography in the courts is a relatively new instrumentality of proof. Consequently, the courts have not yet considered many of the problems involved in the use of such medium. Nevertheless, we are able to ascertain certain definite trends.

We know that, despite the courts' native conservatism, they have been rather liberal in upholding rulings admitting color films and prints in evidence. California, Colorado, Delaware, Idaho, Kansas, Kentucky, Louisiana, Minnesota, Massachusetts, Nebraska, New Jersey, Ohio, Oklahoma, Oregon and Texas, 15 jurisdictions, have sanctioned the use of color photography, with only the State of Montana rejecting such proof.

Color as such does not seem to be the significant factor involved in most of such decisions. If *color* itself is placed in issue, the courts will undoubtedly require a verification showing that a color film or print is a true, fair and correct *color* representation of the person, thing, or scene portrayed (15). So far the courts have not stressed this point too much.

It has been made clear, however, that the general rules applicable heretofore to black and white photographs will also apply to color films (16).

That a color film is gruesome does not render it inadmissible per se where the medium is relevant and material to prove a point in issue. It is only where its inflammatory nature outweighs its relevancy and materiality that courts will exclude color films, as indicated in the ruling of the Montana court.

If color itself is in issue, false rendition of color should affect admissibility of color films. Nevertheless, if it is proved that the colors are approximately correct and representative of the colors of the objects portrayed, slight or

inconsequential deviations from true color values should affect the weight but not the admissibility of color films (17).

The cases generally hold that as a matter of trial practice, the courts will permit color transparencies to be projected on a screen, using a projector for that purpose. That projection will enlarge the image to a considerable degree and thus accentuate the details seems to be of no legal significance (18).

The realism which the color medium portrays cannot be overlooked in the judicial process. The courts should, therefore, continue to deal favorably with color photography. They certainly have not been frightened by the color aspects of color films and prints.

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6. Gold Tone Studios v. Federal Trade Commission, 183 F. (2d) 257, 258 (C. A. 2d 1950).
7. Moon v. State, 68 Ga. 687, 695 (1882), diagram marked with red ink admitted; State v. Dunn, 161 La. 532, 109 So. 56 (1926), writ of error dismissed 273 U. S. 656, 47 S. Ct. 344, 71 L. Ed. 825 (1927), enlargement of palm print, portions being circled in red ink, held admissible; Harris v. Snider, 223 Ala. 94, 134 So. 807 (1931), tinted photograph was admissible to show facial condition before injury. See also: People v. Madison, 3 Cal. (2d) 668, 46 Pac. (2d) 159 (1935); Moore v. Jansen, 265 Ill. App. 459 (1932), where the drawing in of red lanterns was held objectionable.
8. 111 Colo. 390, 142 P. (2d) 277 (1943).
9. Richardson v. Missouri-K-T. R. Co., 205 S. W. (2d) 819 (Tex. Civ. App. 1947).
10. Pruit v. State, 270 P. (2d) 351 (Okla. Cr. App. 1954), body pictures, showing nature of wound; State v. Long, 195 Ore. 81, 244 P. (2d) 1003 (1952), admission of Kodachrome transparency of decedent's head, projected on screen; Armijo v. People, 304 P. (2d) 633 Colo. (1956), color slides of deceased's corpse, showing knife wounds as well as surgeon's incision to save life; State v. McMullan, 223 La. 629, 66 S. (2d) 574 (1953) color transparencies of victim and scene; People v. Moore, 310 P. (2d) 969 (Colo. 1957), color slides of deceased, to show nature of wounds; State v. Kuhnhausen, 201 Ore. 478, 272 P. (2d) 225 (1954), color transparencies of victim and scene. The witness verifying the slides was permitted to set up a projector and project such slides on the screen; State v. Sheppard, 100 Ohio App. 345, 128 N. E. (2d) 471 (1956), affirmed 165 Ohio St. 293, 135 N. E. 340 (1956), cert. denied 352 U. S. 910, 77 S. Ct. 118, 1 L. Ed. (2d) 119 (1956), head of a homicide victim was photographed in color and projected to a 4' x 4' size on a screen; State v. Huff, 14 N. J. 240, 102 A. (2d) 8 (1954), color slides of victim admitted to show nature of wounds. See also State v. Wise, 19 N. J. 59, 98, 115 A. (2d) 62, 82 (1955); State v. Smith, 142 A. (2d) 890 (N. J. 1958); State v. Bucanis, 138 A. (2d) 739 (N. J. 1958); Commonwealth v. Makarewicz, 132 N. E. (2d) 294 (Mass. 1956), colored slides of victim projected on the screen to illustrate testimony of a pathologist; Fields v. State, 284 P. (2d) 442 (Okla. Cr. App. 1955), color slides of victim; Wisniew-

ski v. State, 138 A. (2d) 333 (Del. 1957), color photographs of deceased showing wounds and also incisions resulting from autopsy procedure; People v. LaVerne, 307 P. (2d) 31 (Cal. 1957), colored slides showing pictures of the brain and a cross section thereof. See also Commonwealth of Kentucky, Department of Highways v. Williams, 317 S. W. (2d) 482 (Ky. 1958), colored slides of property.

11. 308 P. (2d) 973 (Mont. 1957); Kiefer v. State, 153 N. E. (2d) 899 (Ind. 1958) seems to approve.
12. State v. Michel, 225 La. 1040, 74 S. (2d) 207 (1954), aff'd 350 U. S. 91, 76 S. Ct. 158, 100 L. Ed. 83, reh. den. 350 U. S. 955, 76 S. Ct. 340, 100 L. Ed. 831; State v. Iverson, 289 P. (2d) 603 (Idaho 1957).
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14. Knox v. City of Granite Falls, 72 N. W. (2d) 67 (Minn. 1955); Anderson v. Evans, 83 N. W. (2d) 59 (Neb. 1957). In Bates v. Newman, 264 P. (2d) 197 (Cal. 1953), it was held that the trial court did not err in refusing to admit in evidence as part of plaintiff's rebuttal 2 color slides of plaintiff's sexual organs. This decision was based on the holding that this should have been part of plaintiff's evidence-in-chief. See also Horowitz v. Bokron, 151 N. E. (2d) 480 (Mass. 1958) and Roring v. Hoggard, 326 P. (2d) 812 (Okla. 1958), color pictures admissible to show extent of injuries.
15. See People v. Moore, 310 P. (2d) 969 (Cal. 1957).
16. State v. Huff, 14 N. J. 240, 102 A. (2d) 8 (1954); Commonwealth v. Makarewicz, 132 N. E. (2d) 294 (Mass. 1956). See also Commonwealth of Kentucky, Department of Highways v. Williams, 317 S. W. (2d) 482 (Ky. 1958), colored slides of property.
17. Green v. City and County of Denver, 111 Colo. 390, 142 P. (2d) 277 (1943), under-exposure considered but held not prejudicial. In People v. Moore, 310 P. (2d) 969 (Cal. 1957), a color transparency had a red or orange effect because of the light and film used, but the court was not too bothered by this fact.
18. In People v. LaVerne, 307 P. (2d) 31 (Cal. 1957), in speaking of transparencies, the court said, "They are *small* pictures." Did the court consider the effect of projection?

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Firearms Discharge Residues*

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The purpose of this investigation was to study the character and distribution of the residues left on surfaces, including human skin, as a result of firearm discharge and to develop a procedure for the chemical qualitative detection of the most frequently encountered metallic or metal-containing components of firearm discharge residues.

Such a test procedure is of major concern to investigators in the field of criminalistics, as it would enable the investigator to determine, with a reasonable degree of certainty, whether or not a suspect had discharged a firearm within a relatively short period of time prior to his apprehension.

The test procedure should be sufficiently simple to be performed by individuals lacking in formal training in chemistry and the results of the test should require a minimum of interpretation. In addition to these limitations, a test procedure suitable for routine use by small as well as large police departments should fulfill the following conditions:

1. Only a small number of inexpensive chemicals should be required.
2. Positive tests should be shown by distinct colors or color changes.
3. The test colors should be stable or capable of being rendered stable.

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4. The time necessary to complete the tests should be short.

There are different types of residues, varying in composition, all of which could be present when a firearm is discharged. The following terms will be used in this report to designate the type of discharge residue under discussion: Firearm discharge residues are residues of powder, primer, lubricants, and metals resulting from the discharge of a firearm. Primer residues are residues resulting from the ignition of the primer only. Leakage residues are residues escaping from a fired weapon from openings in the weapon other than the muzzle.

The first part of the investigation was a study of the qualitative chemical composition of primer and leakage residues and methods for the analysis of the metallic or metal-containing constituents of these residues. It was found that the presence of antimony, barium and lead could be used to characterize all three types of residues. Experimentation proved that one reagent, sodium rhodizonate, was satisfactory for the detection of both barium and lead even in the presence of each other as well as other constituents of firearm discharge residues. The reagent triphenylmethylarsonium iodide was used for the detection of antimony.

The second part of the investigation was a study of the spatial distribution of leakage residues on the hands of persons who had discharged a firearm, and the development of a method of removing this residue for chemical testing. It was found that a small piece of white, cotton cloth moistened with 0.1 normal hydrochloric acid could be used to remove firearm discharge or leakage residues from the hands, and after drying the cloth, chemical tests could be performed on the cloth without further concentration of the residues.

Laboratory tests and use of the developed procedure in actual criminal cases demonstrated that the chemical tests used were specific and sensitive and that the spatial distribution of the leakage residues was of such a consistent pattern as to insure the reliability of the method for determination of the firing of a revolver by a suspect. Thorough or re-

peated washing of the hands prior to the test procedure would probably result in negative findings.

Review of Literature

Composition of Ammunition and Discharge Residues. Detailed information regarding the chemical composition of firearms, fouling, bullets, lubricants, powders, primers, and firearm discharge residues has been furnished by Walker (1). The analysis of ninety-six makes and types of cartridges representing both smokeless and black powder and both new and old ammunition disclosed that the elements or compounds of the elements—antimony, barium, lead, mercury, and potassium—are the most frequently occurring constituents of primers. The older types of primers were more likely to contain mercury and potassium while the newer types had higher concentrations of lead and barium. Antimony was found in both new and old primers.

Hatcher (2) described the chemical composition of a number of different primers, both new and old, as well as furnishing other data on ammunition.

Holmes (3) furnished information regarding modern practices in primer formulation. According to Holmes, "Manufacturers in this country use primers in which the basic explosive ingredient is normal lead styphnate. The explosive is usually lead azide, but the use of mercury fulminate has not been completely discontinued. The oxidizing materials are usually barium nitrate, lead nitrate, or lead peroxide, although other oxidizers may be used. Fuels generally are antimony sulfide, calcium silicide, and powdered aluminum, but recently some companies are introducing powdered zirconium, magnesium, and sometimes titanium into their primers. The sensitizing material may be tetrazine, which is most commonly used, TNT (trinitrotoluene), PETN (pentaerythretol tetanitrate), or others. Abrasive materials are not used in center fire primers but are used in rimfire shells. This material is usually ground glass."

The review of the literature disclosed that the most frequently occurring metallic elements present in primer, leakage, and firearms discharge residues are antimony, barium, and lead. On the basis of this information, the decision was made to review published methods for specific and sensitive tests for these three elements or compounds containing these elements.

Chemical Tests for Antimony, Barium, and Lead

Antimony: The following tests for antimony seemed most promising for further investigation: 9–methyl–2, 3, 7–trihydroxylfluorone (4), Rhodamine B (5), Phosphomolybdic acid (6), Iodide (7), Potassium Iodide and Pyridine (8), and Triphenylmethylarsonium Iodide (9).

Barium: The Sodium Rhodizonate test recommended by Snell and Biffin (10).

Lead: The Sodium Rhodizonate test reported by Feigl (11) and Caldas (12).

The use of 1:20 hydrochloric acid used as an additional step in this test serves not only as a confirmatory test for lead but also as a test to distinguish between barium and lead.

Preliminary Studies on Firearm Discharge Residues. Meyers (13), working on the identification of antimony, barium, and lead in primer residues as part of his senior research at the University of Rhode Island, investigated the use of the contact print method employed by Gutzeit (14) for the detection of metallic constituents in minerals. This method used glossy photographic paper from which the silver salts had been removed. The paper was impregnated with an attacking agent and pressed in contact with the surface to be tested, then developed with a specific reagent. The method was not satisfactory for the removal of leakage residues from the hands.

Whitehead (15), working on the same problem, investigated the removal of leakage residues from the hands by means of pieces of white, cotton cloth moistened with different solvents and found that 1 normal hydrochloric acid was a satisfactory solvent for this purpose. He also investigated procedures for dissolving the residue from the cloth and making chemical tests for antimony, barium, and lead in the resulting solution.

The method of analysis proposed by Whitehead was based on paper-strip chromatography for the separation of antimony, barium, and lead with the identifying test for each element performed as a spot test on the chromatogram. Antimony was identified by means of an iodide test using anti-pyrene in potassium iodide. Barium and lead were identified by means of the Sodium Rhodizonate and 1:20 hydrochloric acid tests.

Whitehead's method was complicated and required considerable analytical technique on the part of the analyst. In addition, the Iodide test for antimony was not specific and barium was not detected in the chromatogram if present in small quantities. The greatest difficulty encountered in the method was the formation of trailing bands formed by lead resulting in a general lack of resolution of the test colors.

Experimentation

Confirmation of Composition of Primer Residues. Spectrochemical procedures were used for determining the presence or absence of antimony, barium, and lead in primer residues obtained from a wide variety of different types and kinds of ammunition. Spectrograms were obtained using a 1.5 meter Applied Research grating spectrograph using d. c. arc excitation with graphite electrodes.

The procedure used to obtain the primer residue was to remove the projectile and the gunpowder from the cartridge case. The cartridge case with its unignited primer was placed in a cleaned revolver of the proper caliber. The revolver was discharged with the muzzle pointed at the center of a small piece of white, cotton cloth placed at a distance of one inch from the muzzle of the revolver.

The cloth bearing the primer residue was placed in a silica crucible and heated in a muffle furnace at 400° C. until a white ash was obtained. The ash was subjected to spectrochemical analysis.

The presence of the elements antimony, barium, and lead in the vast majority of primer residues, as reported in the literature, was confirmed by these experiments.

Chemical Tests for Antimony, Barium, and Lead

Antimony: As the result of experimentation with a number of different chemical tests for antimony the following information was obtained: The Rhodamine B test is relatively insensitive and is not specific. The Phosphomolybdic Acid test is sensitive but it is not specific due to the fact that a number of different oxidizing agents give the same test as antimony. Although 9–methyl 2, 3, 7–trihydroxy–6–fluorone test is sensitive to one part of antimony per 5 million, barium gives the same test; the reagent is unstable; the reagent is not available commercially and its synthesis is difficult. Neither the Iodide nor the Iodide-Pyridine tests are specific for antimony. The Triphenylmethylarsonium Iodide test for the detection of antimony is both sensitive and specific.

The investigation of the above-listed tests for antimony disclosed that the Triphenylmethylarsonium Iodide test was the only test meeting the requirements of sensitivity and specificity. In the presence of trivalent antimony, a 10 per cent alcoholic solution of this reagent produced an orange-colored precipitate in about 30 seconds.

Triphenylmethylarsonium Iodide is not available commercially but it can be synthesized according to the method of Dwyer, Gibson, and Nyhalm (9). The method of preparation of the compound can be simplified by starting with commercially available triphenylarsine, a product in the first step in the synthesis. Triphenylarsine (10 g.) and methyl iodide (10 g.) are dissolved in absolute ethanol (20 ml.) and the solution refluxed for three hours. Dry ether is then added to precipitate the salt triphenylmethylarsonium iodide. Purification is accomplished by redissolving in alcohol and precipitating with ether. A drop or two of sulphurous acid in the alcohol removes the trace of free iodine which tends to form.

Barium: Only one test for barium gave promise of meeting the requirements of specificity, sensitivity, and colored reaction product. In the presence of barium, a 5 per cent solution of sodium rhodizonate produces a red precipitate of barium rhodizonate. Barium rhodizonate was not soluble in 1:20 hydrochloric acid and its red color was not changed by the addition of this acid.

Lead: In the presence of lead, sodium rhodizonate produced a red precipitate of lead rhodizonate. When 1:20 hydrochloric acid was added to red lead rhodizonate the color changed from red to blue. Since 1:20 hydrochloric acid changed the color of lead rhodizonate from red to blue but did not alter the red color of barium rhodizonate, it was possible to use the reagent, sodium rhodizonate, for the detection of both barium and lead in the presence of each other whenever the 1:20 hydrochloric acid test was used.

Specificity of Selected Tests

The specificity tests were made on white, cotton cloth since it was hoped that at least one of the primer, or leakage residue components, could be identified on the cloth used to remove the residue from the surface where it has been deposited.

One molar solution of each of the chemical individuals likely to be present in firearm discharge residues was prepared as test solutions. One drop of each of the test solutions was placed on separate pieces of white, cotton cloth. Each cloth was dried and the test reagent was added. The results of these tests, as listed in Table 1, confirmed the fact that the selected tests are specific in the presence of the components of firearm discharge residues.

Development of an Analytical Procedure

When it had been established that the selected tests for antimony, barium, and lead were specific, several different analytical procedures for the detection of these three elements in the presence of each other were investigated.

The first procedure attempted was based on paper-strip chromatography for the separation of the antimony, barium, and lead. The test reagents were added to the dried chromatogram. It was found that this procedure was not satisfactory because lead formed training bands instead of being separated as a specific band on the paper.

The next approach was to dissolve the residues from the cloth and test the resulting solution. The dilution factor proved too great for sensitive testing and concentration of the solution to improve sensitivity was time-consuming if care was taken to avoid any loss of test material.

It was decided to attempt to identify the three elements by direct testing of the cloth used to remove the residues. Such a procedure would have the advantages of simplicity of procedure, least danger of loss of test material, and a minimum of laboratory equipment. The procedure finally developed was as follows:

1. One drop of a solution containing antimony, barium, and lead was placed on a 2 inch square piece of white, cotton cloth. The cloth was dried and one drop of a 10 per cent alcoholic solution of triphenylmethylarsonium iodide was added. An orange ring indicating the presence of antimony developed in approximately 30 seconds. Full color development of this ring was attained in 2 minutes.
2. The cloth with the orange ring was dried and 2 drops of freshly prepared 5 per cent solution of sodium rhodizonate was added to the center of the orange ring. A red color, showing the presence of barium, lead, or both elements, developed inside the orange ring.

Table 1. Results of Specificity Tests.

ION	Ph_3MeAsI	Na Rhodizonate	1:20 HCl.
Cu^{+}	Pale Yellow	Slight Orange	Clears
Cu^{++}	. . . . .	. . . . .	. . . . .
Cd^{++}	. . . . .	. . . . .	. . . . .
Bi^{++}	. . . . .	. . . . .	. . . . .
Al^{+++}	. . . . .	. . . . .	. . . . .
Hg^{+}	. . . . .	Red Orange	Clears
Hg^{++}	. . . . .	Brown Red	Clears
Fe^{++}	. . . . .	Red Brown	Clears
Fe^{+++}	. . . . .	. . . . .	. . . . .
Pb^{++}	Pale Yellow	Deep Red	Blue
Sn^{++}	. . . . .	Violet	Clears
Sn^{++++}	. . . . .	. . . . .	. . . . .
Sb^{+++}	Orange	. . . . .	. . . . .
Ba^{++}	. . . . .	Red Brown	Red Brown
Zn^{++}	. . . . .	. . . . .	. . . . .
K^{+}	. . . . .	. . . . .	. . . . .
SCN^{-}	. . . . .	. . . . .	. . . . .
NO_3^{-}	. . . . .	. . . . .	. . . . .
$S_2O_3^{=}$	. . . . .	Red	Clears
Ni^{++}	. . . . .	. . . . .	. . . . .
NO_2^{-}	. . . . .	Slight Orange	Clears
$S^{=}$	. . . . .	Black	Clears
$SO_3^{=}$	. . . . .	Orange	Clears
CO_3^{-}	. . . . .	. . . . .	. . . . .

Sodium rhodizonate is decolorized by acids; therefore, it is necessary to remove acids before using this reagent for a test. The cloth must be acid-free before the addition of sodium rhodizonate to assure the maximum intensity of color due to lead and barium rhodizonates. Drying can be accomplished by a hot plate, blast of warm air, radiator, infrared lamp or a hair drier.

3. The cloth from step 2 was dried and 1–2 drops of 1:20 hydrochloric acid were added to the area of the cloth containing the red color. A blue color, which developed inside the orange ring, confirmed the presence of lead. A red color which usually remained in the center of the blue area, confirmed the presence of barium.

It was found that the cloth should be dried after the addition of sodium rhodizonate and before the addition of 1:20 hydrochloric acid. This drying must be done in the absence of strong light because the rhodizonate colors fade in strong light. Blank tests were run on sodium rhodizonate solutions and it was found that strong light decolorized the solutions in a short period of time rendering them incapable of reacting with lead and barium.

The volume of 1:20 hydrochloric acid added should be kept at a minimum. Best color contrast was obtained when only one drop of the acid was used.

Determination of Sensitivity Tests

The sensitivities of the tests using each of the two reagents and the 1:20 hydrochloric acid were not determined until a method of analysis had been developed since it was believed necessary to obtain these data using the same procedure employed in the actual analysis procedure.

Standard solutions containing varying quantities of antimony, barium, and lead in each solution were used for making sensitivity tests. The standard solutions were added to pieces of white, cotton cloth and the analytical procedure previously described was used. The standard solutions were diluted and tested until tests for the presence of antimony, barium, and lead were no longer obtained.

The results obtained were as follows: Four micrograms of antimony were detected in the presence of 1.5 milligrams of lead and 10 milligrams of barium. Ten micrograms of barium were detected in the presence of 1.5 milligrams of lead. It was found that if the concentration of lead is above 1.5 milligrams its presence will mask the test for barium. It was established that 2.5 micrograms of lead can be detected following the identification of antimony and barium.

Stability of Colors

The stability of the colors was determined by storing cloths which had been tested for leakage residue. The cloths stored in the absence of light showed no appreciable color change over a period of three months. The cloths stored in light lost about 50 per cent of the rhodizonate colors during a three-month period. The orange color of the antimony test was not altered by storing in light.

Removal of Discharge Residues from Hands

Several different classes of solvent were tried for complete removal of firearm discharge residues from the hands. These solvents included methyl alcohol, ethyl alcohol, xylol, benzene, phosphoric acid, acetic acid, sulfuric acid, nitric acid, and hydrochloric acid. Hydrochloric acid proved to be the must successful even at concentrations as low as 0.1 molar. An advantage of using a low acid concentration was that it was easier to remove the acid before making the Rhodizonate tests.

The method of removal was to moisten with 0.1 molar hydrochloric acid a piece of white, cotton cloth 2 inches square. The moistened cloth was used for swabbing the areas of the hands being tested for discharge residues.

Analysis of Cloth Used as Swab

The piece of cloth used as a swab was dried and the tests conducted as previously described. In practice, substantial quantities of dirt from the hands were transferred to the swab causing a discoloration of the cloth. A second addition of triphenylmethylarsonium iodide was necessary to wash the orange ring beyond the dirt. The blue color and the red color showing the presence of barium and lead were visible through the dirt. The identification of colors in the presence of dirt offered no difficulty.

Localization of Leakage Residues

A study of the localization of leakage residues originating from the firing of revolvers and semi-automatic pistols disclosed that certain areas of the hand in which the weapon

was held when it was discharged were most likely to be contaminated with leakage residues. When a revolver was fired the pattern of the leakage residue contamination of the hand followed a general pattern with some variation depending upon the size and type of revolver and the degree of gas leakage from the weapon. The firing of a semi-automatic pistol did not result in a general pattern of leakage residue contamination on the hand and, in most instances, a test for leakage residues could not be obtained when a semi-automatic pistol was fired.

The areas of the hands were classified into twelve separate areas as shown in Figure 1. This division was done to enable the investigator to correlate the leakage-residue pattern in the test-firing program. The test procedure used was to test each of the twelve areas of each hand of a person who had fired a weapon.

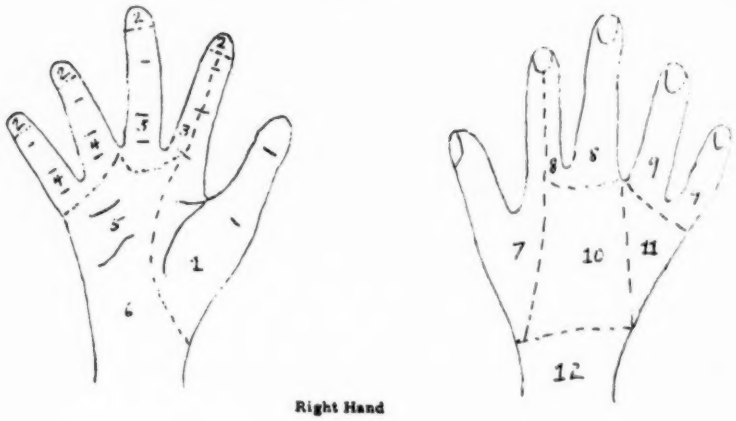
When the hand, which was used to discharge a revolver, was tested it was found that area 7 was the most probable area of greatest concentration of leakage residues and area 1 the next most likely area of contamination. There were slight variations in the location of the leakage residues, depending upon the size and leakage characteristics of each individual revolver, but the general pattern of distribution of leakage residues on the hand holding the revolver was constant. Experimentation disclosed that if a person had handled a recently-fired weapon, but had not actually fired the weapon, leakage residues were most likely to be found in areas 2, 3, 4, and 5.

In all instances where the test procedure was used in actual criminal investigation the weapon involved was fired by an investigator and the hands of the investigator were tested to determine the leakage characteristics of that particular weapon.

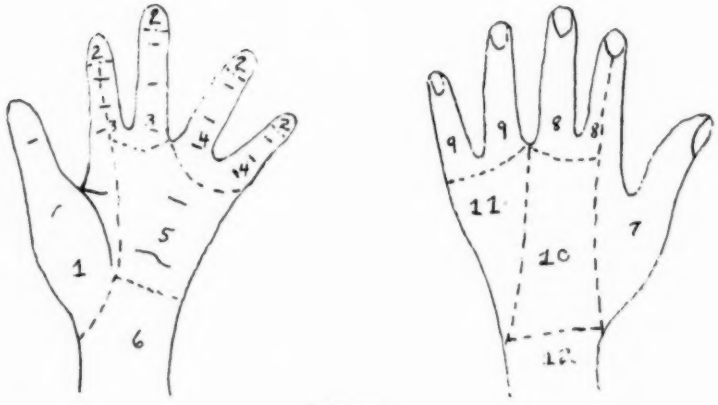
In no case, where a person had fired a revolver, did the chemical tests and localization pattern of leakage-residue contamination fail to disclose that fact. There have been no instances of false positive tests obtained from hands not used to fire a revolver. During the testing program subjects being tested resorted to such deception as wearing gloves, wrapping the hand with a handkerchief or other type of cloth, and dis-

coloring the hand or portions of the hand with graphite powder to simulate the appearance of leakage residues. When there were no leakage residues on the hands the test proce-

Figure I



Right Hand



Left Hand

dure gave negative results. However, when gloves were worn the leakage residues were found on the proper area of the

glove, and when the hand was wrapped in a handkerchief tests were obtained on the handkerchief without removal of the leakage residues.

Testing Program Employing Weapons

Three hundred rounds of ammunition were fired in a large number of different revolvers and semi-automatic pistols. Each weapon was cleaned thoroughly before each firing. Two hundred and fifty of the rounds were fired from 22-caliber weapons using a variety of ammunition including Remington and Western Super X, manufactured by Olin Industries; Winchester, manufactured by the Winchester Arms Co.; Sta-Klean, manufactured or distributed by Sears Roebuck Co.; Peter's, manufactured by the Peters Ammunition Co.; United States, manufactured by United States Ammunition Co. Fifty rounds of ammunition were fired from a variety of revolvers and semi-automatic pistols available to these laboratories and some revolvers reported to have a minimum of gas leakage which were brought to the laboratories for testing purposes.

Each time a weapon was fired the hands of the person who discharged the weapon were tested by the procedure previously described and all cloths used for chemical testing were ashed and spectrochemical analyses made to check the chemical tests. Whenever revolvers were fired the presence of lead was detected in all tests made during the testing program. In a few instances, where antimony or barium was not found by chemical tests, these elements were not found in the spectrochemical analysis of the same cloth used in the chemical test. In no case where antimony, barium and lead were present in the primer did the chemical test fail to show the presence of these elements in the leakage residues. In every case where a revolver type weapon was discharged positive results were obtained from at least one of the cloth swabs used in wiping the hands of the person who had fired the weapon.

Semi-automatic pistols seldom leak sufficient quantities of residues to give positive tests. A few semi-automatic pistols did have sufficient gas leakage to give positive results but this is the exception rather than the rule.

Tests for Interferences

A series of tests were performed on persons involved in occupations where occupational contamination of the hands might cause interference with the test procedure either by lack of sensitivity of tests or false tests due to lack of specificity of tests employed. Persons whose hands were contaminated with such substances as fertilizer, photographic chemicals, metal finishing chemicals, plumbers' supplies, costume jewelry, tobacco, tobacco smoke, tobacco juice, urine, saliva, firecrackers, cosmetics, and various bleaching agents were tested before and after firing a revolver. In no instance was difficulty encountered in determining the fact that a revolver had been fired and which hand had been used to discharge the revolver. Tests for antimony, barium, and lead were positive and the location of the leakage residues was consistent with firing a revolver and not occupational contaminations.

Effect of Washing Hands and Time Factor

A number of tests were performed on the hands of persons who had fired a revolver and then washed their hands before being tested. In some instances leakage residues could not be detected on the hands after the hands had been washed thoroughly once. When extreme care had not been taken to wash the hands as thoroughly as possible it was possible to obtain positive tests after two or three casual washings. However, experimentation indicates that firearms discharged residues can be removed from the hands after one washing if the washing is done thoroughly.

When unwashed hands were tested it was possible to detect the presence of leakage residues for a period as long as 48 hours after firing a revolver.

Use of Test Procedure in Actual Cases

Case 1: Our laboratories were requested to assist a police department in the investigation of a murder involving the death of a woman who was believed to have been shot in the back by her sixteen-year-old son. It was specifically requested that the boy's hands be tested to determine if he had fired a 22-caliber semi-automatic pistol. The boy had been apprehended immediately after the shooting had occurred and had not been allowed to wash his hands. Tests, as previously described, were made on the boy's hands

and strong positive tests for antimony, barium, and lead were found on the pieces of cloth used to swab the inside surface of the left hand but tests for these elements were negative for the areas of the right hand.

The shooting had taken place in an automobile trailer home and four ejected but unfired 22-caliber cartridges and three 22-caliber shell casings were found on the floor of the trailer in the room where the shooting had occurred. When the boy was informed that the police knew that he had handled a recently-fired weapon he confessed to the shooting. He stated that he did not understand the operation of a semi-automatic pistol and that he thought it was necessary to cock the pistol by hand moving the slide every time the pistol was fired. Therefore, after each shot he had placed his left hand over the slide and ejected a live cartridge and contaminated the inside of his left hand with leakage residues left on the slide each time the pistol was discharged. The results of the tests for the presence of leakage residues were introduced in court during the trial which resulted in a conviction.

Case 2: A young man was fatally wounded during a family argument. When the police arrived at the scene the victim's brother and sister-in-law were present. The victim's brother told the police that the revolver used in the shooting was in the bushes near the front door and it was recovered from that place by the police. The police requested our laboratories to test the hands of the victim's brother and sister-in-law to determine if either had fired a revolver.

The tests disclosed that the brother had leakage residues on the tips of the fingers of the right hand but no leakage residues on areas 7 or 1. The victim's sister-in-law had leakage residues on the tips of the fingers of both hands. Prior to the tests, each had admitted doing the shooting and had then accused the other, but as a result of the tests, which showed that neither had fired the revolver, they admitted that a second brother had been present at the time of the shooting but had fled the house before the police arrived.

The missing brother was apprehended several hours later and brought to the police station for testing. The results of the tests showed the presence of leakage residues on the subjects right hand in the same location as those found on the right hand of an investigator who had made a test shot with the revolver used in the shooting. When confronted with this information the second brother confessed to the shooting and claimed self defense.

Suicides: The test procedure has been used in the investigation of a number of cases of suicide by shooting with a revolver and in each case the results of the tests were in agreement with other available evidence.

Other Applications: A modification of the test, involving the test for lead only, has been used on numerous occasions to determine whether or not certain holes were made by bullets, identification of bullet marks on stones and sinter-block walls, and tests on patches used in the "patch test" to determine if a weapon has been fired since cleaning.

Summary

1. The procedure for the detection of firearm discharge residues employs three chemical reagents only. The tests are

colorimetric and the technique is sufficiently simple to eliminate the necessity of technical training as a prerequisite for performing the tests. The results of the tests are shown as definite colors on a piece of cloth and the colors are stable over a considerable period of time.

2. Positive results were obtained in all cases in which revolver-type weapons were used. In a few instances positive tests were obtained when semi-automatic pistols were used. However, it is necessary that a weapon leak gases and residue in order to deposit discharge residues on the hands.

3. No false tests were obtained nor failure of tests to detect antimony, barium, and lead were encountered because of occupational contamination of the hands.

4. There was excellent correlation between the chemical tests for firearms discharge residues and spectrochemical tests of the same swabs.

5. Detection of Firearm Discharge Residues: Proposed Procedure.

(1) Removal of Residues: Swab surface with 2-inch square piece of white, cotton cloth moistened with 0.1 molar hydrochloric acid.

(2) Detection of Antimony: Dry swab used to rub area to be tested. Add 1 or 2 drops of 10 per cent alcoholic solution of triphenylmethylarsonium iodide. The appearance of orange ring on cloth is a positive test for antimony. Two minutes should be allowed for full color development of ring.

(3) Detection of Barium and Lead: Dry cloth used in antimony test. Add 2 drops of freshly prepared 5 per cent solution of sodium rhodizonate to center of orange ring. Appearance of red color inside of orange ring is positive test for barium, or lead, or both barium and lead.

(4) Confirmation of Test for Barium and Lead: Dry cloth from (3) with drying accomplished in absence of strong light. Add 1–2 drops of 1:20 hydrochloric acid to red-colored area. A blue color developing inside of orange ring is confirmation of test for lead. A red color

usually remaining in center of orange ring is confirmation of test for barium.

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Diffuse Injury to the Respiratory Tract from Intratracheal Blast Due to Ether Explosion*

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Of the considerable information in the literature (1–6) on bodily injury from explosion, relatively little relates to shock wave in the trachea from explosion of anaesthetic. Since present consensus favors the chest wall rather than the bronchial tree as the medium conducting shock wave from external explosion to the lungs, it would be of some interest to note whether the pathologic picture of intratracheal blast from anaesthetic explosion differs significantly from the pattern in bomb or other external explosion. Fortunately the material on anaesthetic explosion is not voluminous, and few of us will be so unfortunate as to see more than one case. It is hoped that those who have relevant observations will present them for comparison and contrast with those recorded here.

Prior to this experience one of us (RWH) dwelt in an ignorance of explosive injury which was almost total. He had tended to the inarticulate assumption that such injury followed an all or none law; that the body was either blown to pieces or uninjured. He was therefore interested and sur-

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prised to learn from the literature that after exposure to explosion blast the onset of symptoms may be delayed for hours and fatality for days, that those dying of blast injury may show no damage to the body surface, and that the pulmonary hemorrhage, edema, and emphysema which are often the principal necropsy finding, may have nothing in their pattern to distinguish them from pulmonary hemorrhage, edema, and emphysema due to other causes.

Case Report

J. A. T., a white male born in 1874, had had resection of the left maxima and left suprahyoid dissection for intraoral carcinoma in 1946. He returned to the emergency room on January 4, 1957 requesting a mouthwash. Examination showed evidence of the previous surgery and a raised ulcerating lesion measuring about 5 cms. on the right lower alveolar ridge. A biopsy showed squamous cell carcinoma. X-ray revealed a large rarified area in the right mandible. From his age, emaciation, and probable senile emphysema he was considered a poor surgical risk, although his cardiovascular status seemed relatively favorable for his age.

Surgery was done February 1, 1957. Anaesthesia was started with pentothal and oxygen, and later oxygen with nitrous oxide were given through a nasotracheal tube in a closed rebreathing circuit. The anaesthetist felt it necessary to supplement further with ether. No odor of ether was noted and the mouth was carefully packed. Unaware that ether had been added, the surgeon continued to use the Bovie cautery. An explosion occurred about two hours after the beginning of the operation. There was a loud roar and a sheet of flame, and the glass ether chamber was shattered. An observer, scrub-suited but not gowned, received second degree burns. Though momentarily stunned, the anaesthetist managed to get the machine out of the way and extinguish the fire on the drapes. As there was no significant fall in blood pressure, the patient was redraped, and the surgery completed. A small amount of blood oozed around the anaesthesia tube. The patient did not recover consciousness after surgery. About 6 hours after the explosion it was noted that there was moderate bloody drainage. Blood pressure was maintained and the chest seemed clear anteriorly. However x-ray showed patchy edema throughout the inner half of the lung fields. The patient died rather abruptly about 11 hours after the explosion. Post-mortem x-ray film showed diffuse granular shadow throughout both lung fields in addition to the edema of the inner lung zones noted in the previous film.

Autopsy was performed 12 hours post-mortem by Dr. Eaton. The body was that of an elderly white male, height 65 inches, weight

estimated at 110 lbs. There was evidence of extensive recent surgery of the neck and jaw. There were no burns on the body surface. There was some air in the tissues of the neck. The chest showed separation of the right ninth and tenth costochondral junctions, without observable reaction in adjacent tissues. We have wondered whether it might have been due, in part at least, to post-mortem attempts at

Fig. 1—Larynx and trachea.

artificial respiration. The 4th rib was fractured through the bony portion, about 4 cms. from the costochondral junction, and there was hemorrhage beneath the parietal pleura in this area. This fracture seemed definitely attributable to the explosion. Considerable serosanguinous fluid was present in both pleural cavities, and several

ecchymotic areas were noted in the parietal pleura. Examination of the airways showed hemorrhage and edema in the ethmoid sinus, pharynx, and bronchi. The bronchi were filled with frothy blood. Hemorrhage was particularly massive beneath the mucosa of the larynx and upper trachea (Figure 1). Hemorrhagic foci were also present in the oesophagus and perioesophageal tissues and in the adventitia of the aorta (Figure 2). No changes suggestive of charring were seen anywhere.

Fig. 2—Oesophagus and aorta.

The right lung weighed 1040 grams and the left 790 grams. Both appeared diffusely congested and hemorrhagic with some emphysema. The brain weighed 1170 grams and appeared moderately congested. There was moderate selerosis of the base of the aorta, and relatively scant selerosis of the coronary arteries which were everywhere patent. The heart weighed 440 grams. The remainder of the gross examination was unremarkable. Microscopic sections showed hemorrhage, edema, and areas of leukocytic exudate in the trachea and larynx, extensive patchy edema and hemorrhage with alveolar dilatation in the lung, submucosal hemorrhage in the oesophagus, adventitial hemorrhage in the aorta, congestion of the brain, and scant to moderate patchy fibrosis in the myocardium (Figures 3–6).

Discussion

Ether was being administered at the time of the explosion and had been started shortly before. Recollections as to whether the cautery was on or off at the moment were a little vague. However, the most reasonable reconstruction of the explosion seems to be that of the fire marshal, that diffusion

Fig. 3—Microscopic section of trachea showing hemorrhage beneath the mucosa.

from the patient's tissues or (possibly) a small leak in the apparatus brought enough ether into the neighborhood of the cautery to serve as a sort of wick which in turn ignited ether in the tube, and that this fire in its turn led to explosion in the glass ether chamber.

If one were to attempt the exercise of reconstructing pathogenesis from post-mortem findings alone without benefit of history, the combination of major ecchymosis in the larynx and trachea with the changes in the pharynx and bronchi, chest wall and the mediastinum would certainly suggest a shock wave within the airway. Indeed, it is difficult to imagine

Fig. 4—Perioesophageal hemorrhage.

a plausible alternative explanation. Conversely, while those of us who were present in the operating room shortly after the explosion were encouraged by the absence of acute fall in blood pressure to indulge in the hope that somehow or other the blast wave may have been damped or detoured so as to minimize "internal bruise," it is hard to hypothecate a means by which such damping or detouring could actually

occur. Thus putting the history and the pattern of laryngo-tracheo-pharyngo-bronchial-mediastinal-chest wall injury together, it was impossible to avoid the conclusion that the airway had sustained a forceful expansile thrust.

In attempting to decide how and why the man actually died, one could give no more weight to the striking complex just listed than to the rather nondescript lesion of the pulmonary parenchyma. The morphology of this lesion is compat-

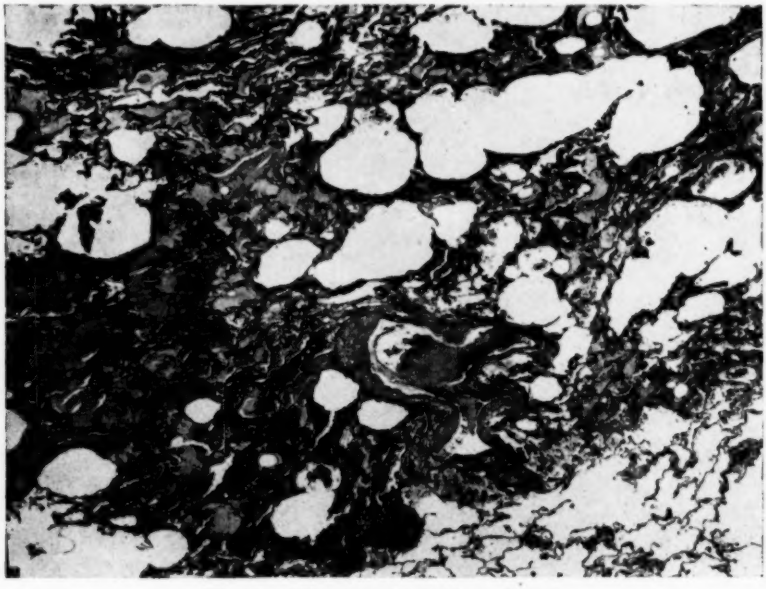

Fig. 5—Microscopic section of lung.

ible with blast injury, but also with a good many other things. Still, the history and the associated lesions force one to conclude that the pulmonary lesion was at least in part attributable to the intratracheal blast. One can speculate endlessly, but to little profit, as to whether a younger and healthier man might have survived an equivalent internal impact. The hypothesis that the man died of a heart-failure lung only slightly influenced by the novel trauma is one which we felt forced to reject. The notion that an explosion death would

have to be instantaneous finds no support in the literature. Conversely, it is a little difficult to understand how a really tottering heart could have kept beating so long following such an insult. Anatomic examination of the heart failed to refute the clinico-physiologic impression of its relative adequacy.

Laryngo-tracheo-mediastinal lesions such as this do not appear to have been recorded as a usual finding in fatality

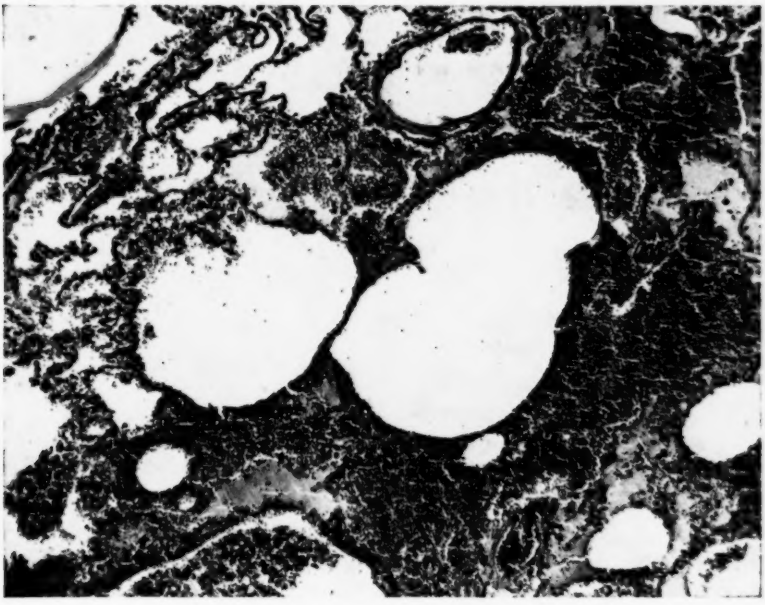

Fig. 6—Microscopic section of lung.

from external explosion. One cannot, perhaps, exclude the possibility that they might occasionally be ignored because of their uncertain lethal significance.

This case suggests that pulmonary lesions from internal blast and from external explosion may have much in common with each other and with those of vehicular trauma. Circumscribed traumatic hemorrhagic lesions of the lungs may

perhaps be designated as contusions; one wonders whether to apply the term to unmarginated lesions like the present. We have been impressed with the occurrence of diffuse pulmonary hemorrhage, edema, and emphysema in individuals dying several hours or more after vehicular misadventure without evidence of major brain damage or of pre-existing heart disease, and with or without rib fractures and pulmonary laceration. We would suggest that such lesions may be of substantial importance as a cause of death following vehicular trauma, and that their pathogenesis presents problems similar to those of blast injury of the lung. One is tempted to speculate on what happens in a chest impact with a closed glottis.

Summary

A case is presented in which death occurred approximately 11 hours after an intratracheal blast from ether explosion. The principal findings were ecchymosis of the larynx, trachea, pharynx, ethmoid sinus, bronchi, and parietal pleura, rib fracture, and a diffuse pulmonary process exhibiting hemorrhage, edema, and alveolar dilatation. The findings are compared and contrasted with those described in external air blast injury and those noted in acute vehicular deceleration.

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Multiple Forgeries*

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Problems are submitted to document examiners from time to time involving multiple forgeries. Such forgeries occur as signatures on face or endorsements on back of checks, or both. The problem usually consists of three parts: (1) whether the signatures and/or endorsements are actually forgeries; (2) if forgeries, were they committed by one or more than one individual; and, (3) who committed the forgeries?

Forged checks drawn on depositors' accounts or checks drawn on fictitious accounts will contain handwritten signatures and endorsements, and the bodies of the checks consisting of dates, names of payees, and amounts may be handwritten or typewritten. It is necessary to pay attention to whether or not more than one person is responsible for preparing the checks, writing the signatures on face and the endorsements on back. In most cases only one individual is responsible but instances are known where two or more persons have conspired to commit such frauds.

A person intent upon committing a forgery or series of forgeries may come into possession of a genuine check written and signed by the maker and use this as a model. When he has a model he usually attempts to copy the handwriting and signatures. If he does not have a model he will probably attempt to disguise his writing. Tracing is rare among these types of forgeries.

Assuming that the examiner has several checks bearing alleged forged signatures and/or endorsements, what should he do? Unless the circumstances clearly indicate the signatures or endorsements are forgeries, he should first establish this by

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comparison with genuine signatures. Having determined that the signatures or endorsements are forgeries, examination and study should be made to determine whether one or two or more persons are responsible. When any one person writes a series of names as signatures to checks or endorsements on backs of checks, even though attempting to copy or disguise, close study will often reveal similar habits or peculiarities linking one individual as the forger. These telltale habits may consist of a number of things such as positioning the writing in relation to the margins, writing lines, slanting the names up or down, peculiarities in certain letters, use or omission of punctuation marks following initials or at the end of names, et cetera.

Sometimes the handwriting evidence is stronger pointing to the identity of the forger when multiple forgeries are involved, rather than if there are only one or two. This is often true even when the forgeries consist of different names. There are cases involving a series of forged signatures or endorsements where the evidence might not be strong enough to identify the forger if the examination was limited to one or two signatures or endorsements, but taken as a whole, positive identification of the forger of the entire group is possible. This statement might not be understood by laymen, including some lawyers, but it is nevertheless true. The reasoning may be better understood when applied to the identification of the writer of a letter or other extended handwritten document. If the examiner were furnished only a few words taken from a lengthy document and asked to make comparison with the writing of a suspect he might not be able to reach a conclusion. But if furnished the entire document for comparison the evidence may be overwhelming. The identification would be based upon a combination of a great number of individual habits occurring throughout the entire writing.

When the evidence indicates that a number of similar offences have been committed by one person or a group of persons, prosecuting attorneys are inclined to limit charges in an indictment to include only one or a few of the overt acts. The report of the examiner should state clearly the basis for his findings and specifically include a description of all documents which would be necessary to support his conclusions

should he later be called upon to testify. Otherwise he may find upon being called into court that his testimony will be confined to one or a limited few of the total number of forgeries he examined and upon which his conclusions were based. He might be "out on a limb" so to speak in his attempt to justify his opinion that the defendant executed the forgeries. The same situation might arise in event all of the standards used for comparison were not received in evidence for lack of identification. If he has stated his position clearly in advance he is in the clear should such a situation arise.

During the past several months this examiner has had submitted to him two cases, each involving a large number of forged checks. In each instance the checks were drawn against the account of a dealer in used automobiles and the forgeries were committed by the bookkeeper for the concern. In one instance more than 200 forged checks were discovered, totaling more than $30,000, and these forgeries occurred over a period of more than two years. In the second case there were sixty odd forged checks, totaling more than $60,000, and these forgeries occurred over a period of more than three years.

In the first case, the bookkeeper, a woman, drew many of the checks payable to her employer, many payable to herself, some to finance companies with which the dealer had accounts, and to numerous other individuals and concerns. She would forge her employer's signatures and endorsements of names of persons or concerns to whom the checks were payable. Some of the checks were cashed by her at the bank in which the dealer carried his account. Many of the checks were run through the account of the dealer and many of them bore the endorsement of a delicatessen owned and operated by the bookkeeper and her husband.

This woman became a very clever forger. It was not difficult to detect her forgeries in the beginning but as she continued she became more proficient and toward the end of her career the forgeries could hardly be detected from the genuine. In fact it is doubtful that all of the forgeries committed by her were detected. When confronted with the evidence, this woman confessed and the matter is now in litigation in an effort to recover the money.

In the second case, the bookkeeper, a man, drew checks against his employer, forged his employer's signatures, and cashed the checks in the bank in which the automobile dealer

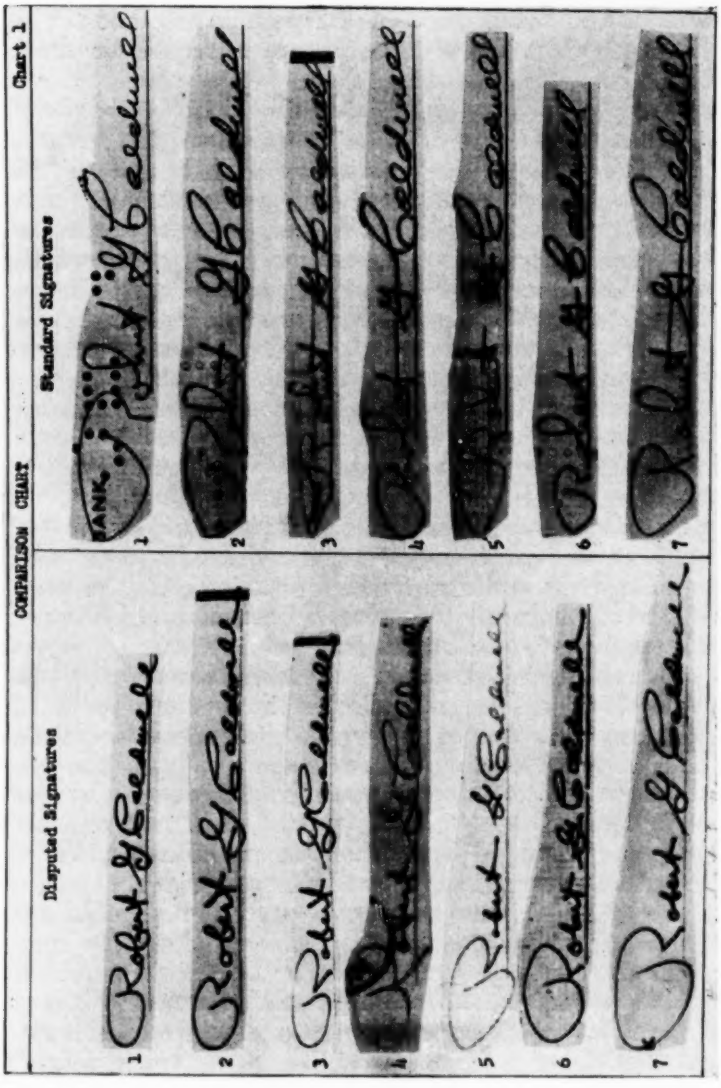

Chart I—Signatures on the left are from microfilm copies of forged checks. Signatures on the right are photographs of signatures of Robert G. Caldwell.

carried his account. When the matter was presented for examination it was explained that none of the alleged forged checks were available and it was presumed the bookkeeper

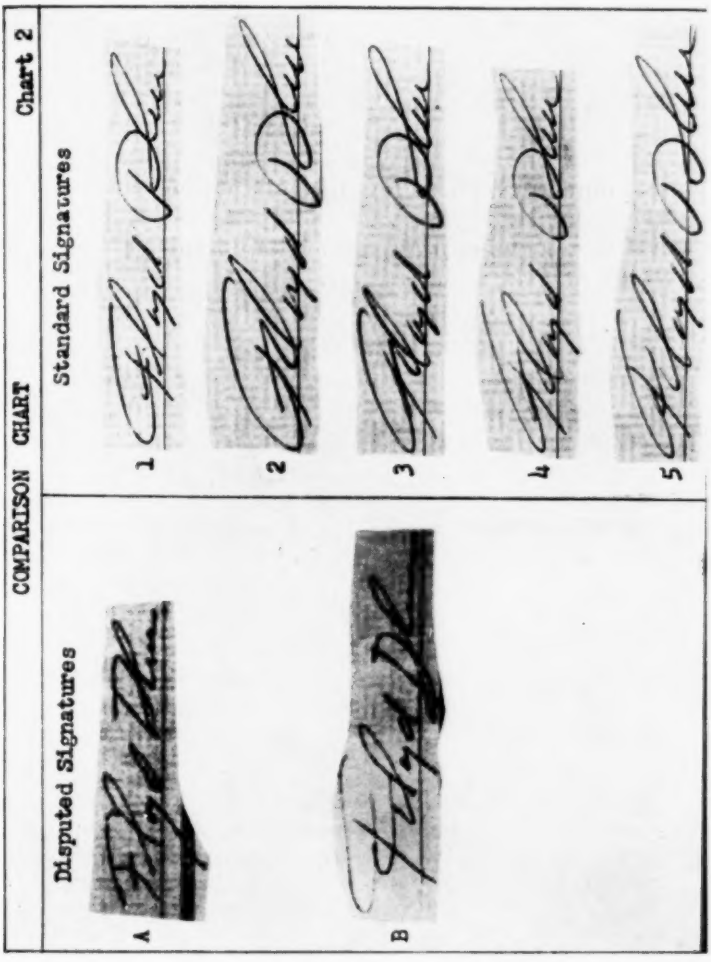

Chart II—Signatures on the left are from microfilm copies of forged checks. Signatures on the right are photographs of the signatures of Floyd Blue.

had removed the checks when they were returned from the bank. Reproduced copies of the checks made from microfilm were furnished. These copies were enlarged to the approxi-

mate size of the original checks. Having had the previous experience with the clever woman forger, the task of examining these microfilm copies was undertaken with some skepticism as to the final results. It developed, however, that the forger in this instance was not a clever forger at all. Although he made an attempt to copy his employer's signatures, the results were crude as evidenced by the accompanying photographs, charts I and II, showing side by side some of the forged signatures with genuine signatures of Robert G. Caldwell and Floyd Blue, the employers. The bookkeeper admitted his forgeries, entered a plea of guilty to the charge of embezzlement, and the bank has made an adjustment. The bank offered a settlement during the trial of a civil suit to recover the money paid on forged signatures.

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Sudden Death Due to Pulmonary Fat Embolism in Chronic Alcoholics with Fatty Livers*

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Sudden and unexpected death is common in individuals with chronic alcoholism associated with fatty livers. Indeed, at autopsy in many instances, replacement of the liver parenchyma by fat is the only anatomic lesion of significance. The mechanism responsible for death in such cases has not been adequately clarified. LeCount and Singer (5), who first called attention to the association of fatty liver and sudden death, attributed death to diminution of the glycogen storing capacity of the liver. Graham (2) analyzed five cases and considered hypoglycemia responsible for the terminal coma, with or without convulsions, and death; although he found no chemical corroboration. Sudden death in animals fed diets deficient in certain vitamins suggested to Graham that a nutritional alteration may be involved.

In choline deficient rats with fatty livers, Hartroft and Ridout (4) demonstrated escape of lipid from hepatic cysts into bile canaliculi and hepatic sinusoids and found fat emboli in the lungs, myocardium and kidneys. The following studies were conducted to determine whether or not pulmonary fat

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embolism might be a factor in sudden and unexpected death in chronic alcoholics with fatty livers. (1)

Methods

Twenty-five cases of sudden death of chronic alcoholics were studied at autopsy. Only those cases without significant anatomical or pathological changes other than fatty liver were included. Those cases with evidence of trauma to soft tissues or bones were excluded. Ten cases of sudden death without evidence of fatty liver or trauma served as controls. Twenty cases with portal cirrhosis were also studied. Fat stains with Sudan IV of frozen sections from the lungs, kidneys, liver, brain and heart, when available, were examined in all cases. In addition, routine hematoxylin and eosin preparations were examined.

Report of Cases

Case 1: 67-year-old white male—A–51–378 (HM).

The deceased was admitted to University Hospital, Baltimore, at 10:30 a. m. and died at 3:45 p. m. He had been reported missing, and was picked up by police in a confused and disoriented state. On admission it was stated by the admitting physician that the patient was incoherent and no real history could be obtained. Physical examination was negative, except for tremors, and a diagnosis of delirium tremens was made. An apparently more acute case was admitted shortly afterwards and the patient was put in another room. When the physician returned a short time later the patient was dead. It was believed by the clinicians that the patient had a coronary occlusion. Past history revealed that the patient was employed part time as a hotel clerk and drank to excess. One year prior to admission he had been beaten, but had not required hospitalization. As far as the employer knew there was no history of trauma immediately prior to admission, nor had the patient been sick.

Autopsy: The body was that of an elderly white male. It was dirty and extensively soiled by fecal material. Rigor and livor were fully developed. There was no external evidence of injury. Brain: There was a very small amount of old blood pigment on the upper surface of the tentorium on the right side, but there was no evidence of contusion of the brain in this region. There were no fractures of the skull. The abdominal organs were in their normal anatomic relationships. The liver weighed 1850 grams. Cut surface was a yellowish color and of a somewhat flabby consistency. The gallbladder, pancreas, adrenals and esophagus were not remarkable. The stomach contained about 400 ml. of coffee ground-like material. The gastric wall showed post-mortem autolytic changes. The small intestine contained opaque material throughout, but this was bile stained. The colon contained liquid, foul-

smelling, dark-brownish material, quite obviously having a large content of blood. Both kidneys weighed 350 grams. The capsules stripped with ease and the sectioned parenchyma showed marked congestion, but was otherwise not remarkable. The lower genito-urinary tract was not remarkable. The combined weight of the lungs was 900 grams. The airways were clear. Sections of the parenchyma were light and air-containing in all portions. The heart weighed 450 grams. Sections of the coronaries revealed only a minimal amount of sclerosis. The valves, chambers and myocardium showed no abnormalities. Blood alcohol determination was negative.

Microscopic: Sections of the lung from multiple areas, stained with Sudan IV, showed many distinct sudanophilic globules within the pulmonary arteriolar and capillary lumens. No emboli were detected in Sudan-stained sections of brain and kidney. The liver showed almost complete replacement of the hepatic parenchyma by fat, which was largely in the form of large, thin walled cysts of estimated diameter 25–100 micra, mostly extracellular.

Case 2: 45-year-old white male—D–53–216 (A.S.).

The history revealed a chronic alcoholic for a period of years, who was found dead in the doorway of his residence. He apparently had been sitting on the front steps and toppled over, falling several short steps. No medical history was obtained.

Autopsy: The body was that of a poorly nourished and poorly kept middle-aged male. There were abrasions across the bridge of the nose, on the left forehead and above the left upper eyelid. There were also abrasions of the skin of the left knee. These abrasions were superficial and measured about 1 cm. in diameter each, and were associated with a small amount of subcutaneous hemorrhage. The chest organs were in their normal anatomic position. The liver was quite yellow in color. The lungs were emphysematous and met in the midline. The heart was small and weighed an estimated 300 grams. Except for petechial hemorrhages on the epicardial surface, the heart was not remarkable. The liver was yellow in color and, on section, was flabby and greasy. The remaining abdominal viscera presented no abnormalities. There was no evidence of extra or intra-cerebral hemorrhage or contusion of the brain. The blood alcohol was 0.22%.

Microscopic: The findings in lung, liver, brain and kidney were identical with Case 1.

Case 3: 61-year-old white male—51–437 (J.K.).

The deceased was attending an Army Engineers Convention in a Baltimore hotel. A friend tried to get into his room at 8:00 a. m. one morning, but was unable to do so, although he heard a shower running. He returned to the room at 9:50 a. m. and found the door open. At this time he found the deceased lying in the bathroom with part of his body in the shower, with the water running. He was lying face down, but it was recognized at the time that he couldn't have drowned because the water was running out the drain in the shower stall. The deceased was scalded from the hot water and still had his shoes and pants on. He was last seen alive by a friend at 1:00 a. m. of the same morning. There were no medicines in the room, and no history of illness prior to time of death was obtained.

Autopsy: The body was that of a well-developed, well-nourished, white male showing no external signs of trauma or violence except for scalding, i. e. 1st degree burns, of the face, arms and thorax. The organs of the chest and abdomen were in their normal anatomic relationship. The serosal surfaces were not remarkable. The heart weighed 340 grams. There were no gross abnormalities of the epicardium, myocardium, valves or coronary arteries. The lungs together weighed 900 grams. They showed a moderate amount of congestion, but otherwise were not remarkable. The liver weighed 1900 grams. Multiple sections revealed the parenchyma to be yellowish-brown in color and greasy in consistency, with almost complete obliteration of the lobular pattern. The gallbladder, biliary tract and pancreas were not remarkable. The spleen was slightly enlarged, weighing 250 grams and, on section, appeared congested but otherwise not remarkable. No abnormalities were noted throughout the esophagus, stomach, and small and large intestines. The kidneys together weighed 240 grams and on multiple sectioning showed no abnormalities. Cisternal puncture revealed a clear spinal fluid containing 0.30% alcohol.

Microscopic: The microscopic findings were identical with those in Cases 1 and 2.

Case 4: 60-year-old white male—53–101 (R.F.).

The deceased was a news vendor and was found dead in his room by a policeman at 12:05 a. m. He was last seen alive by friends at 9:00 a. m. the previous morning. He was a known chronic alcoholic and, when found, wine bottles were scattered over his room. There was no previous medical history.

Autopsy: The body was that of a fairly well-developed and well-nourished, white male weighing 140 lbs. There were no external marks of injury or trauma on the body. The abdominal organs were in their respective anatomic locations. The liver was markedly enlarged, weighing 5000 grams. It was yellow in color and, on section, was greasy, and fat could be expressed upon pressure. The spleen was not remarkable. The gastro-intestinal tract presented no abnormalities. The kidneys showed a diffuse fine dimpling, but were otherwise not remarkable. The ureters and bladder were not remarkable. The lungs were crepitant throughout and showed no abnormalities. The heart was small. The musculature presented a brownish discoloration and was friable. The coronary arteries were patent and showed minimal sclerosis. The aorta showed minimal arteriosclerosis. The blood alcohol was 0.30%, the spinal fluid alcohol 0.29%.

Microscopic: Microscopic sections showed marked pulmonary fat embolism and fatty liver change identical to that described in the other cases. Sudan-stained sections of the brain and kidneys were negative.

Case 5: 32-year-old colored male—53–579 (W.J.).

The deceased was found by his landlady in an attack of acute respiratory distress with subsequent death almost immediately. In his room an empty gin bottle was found. He was a known chronic alcoholic. No medical history.

Autopsy: The body was that of a well developed, well nourished, young, colored male. There were no external marks of injury. Rigor and livor were

not present and body heat seemed extremely high. There was no evidence of injury to the scalp, brain or skull and the spinal fluid was crystal clear. Multiple sections of the brain showed no gross lesions. The heart weighed 320 grams. The coronary arteries were widely patent. The myocardium was somewhat soft and flabby and tan in color. The valves and chambers were not remarkable. The lungs weighed 1020 grams together. The airways were patent. Sections showed noticeable petechial hemorrhages through the substance and there was a moderate amount of dependent edema. The liver weighed 2420 grams. Sections were of a light tan color and greasy. There was no increase in fibrous tissue. The liver capsule was tense and, on section, the edges bulged. The spleen weighed 160 grams and the cut surface was dry. The pancreas, adrenals and kidneys showed no abnormalities. The esophagus was not remarkable. The stomach contained about 200 ml. of liquid fluid with a distinctive alcoholic odor. The small and large intestines were not remarkable. The spinal fluid alcohol was 0.11%.

Microscopic: The findings were similar to the cases described above.

Case 6: 27-year-old colored male—53–609 (H.M.).

The deceased was a heavy wine drinker and known by the police for a number of years. He was arrested for drunkenness and taken to a district jail at 7:10 a. m. one morning. At 7:30 a. m., same morning, according to his cell mate, the deceased rolled out of his bunk and hit his head on the floor. He was pronounced dead at 8:00 a. m. No medical history was obtained.

Autopsy: The body was that of a poorly kept, colored male. In addition to extreme filth, the odor of neglect so characteristic of a chronic alcoholic was apparent over the body. The only external evidence of injury was a superficial contusion of the ribs and of the left lateral region of the mouth. The brain showed no abnormalities on surface or on cut section. The base of the skull was intact. The meninges were not remarkable. The neck organs were explored in situ and no abnormalities were noted. The heart weighed 260 grams. The coronary arteries were sclerotic but no obstruction was noted. The myocardium was flabby and tan in color. The valves and chambers were not remarkable. There was no scarring or necrosis of the myocardium. The lungs together weighed 850 grams. There were multiple adhesions over the surface of the left lung which bound the visceral to the parietal pleura. The airways were patent. Multiple sections of the lungs showed some hyperemia and edema in the lower lobes, but no evidence of consolidation. There were multiple, large, peribronchial calcified lymph nodes. The liver weighed 2150 grams. On sectioning the stroma was a light tan in color and greasy to touch. The spleen weighed 350 grams and was congested. The gastro-intestinal tract was not remarkable. The kidneys weighed 460 grams together. The capsules stripped with ease. Sections showed no abnormalities. The pancreas and adrenals were not remarkable. The spinal fluid alcohol was negative.

Microscopic: Multiple sections of the lung stained with Sudan IV showed numerous fat emboli occluding the lumens of the pulmonary arterioles and capillaries. The majority of the emboli were large, measuring 25–40 micra, approximately. H and E sections of the lung revealed a healed primary tuberculous complex. Sections of the liver revealed marked distortion of the

architectural pattern by massive fatty change, consisting largely of extra cellular cysts. Fat stains of kidneys or brain were not made due to inadvertent discarding of the wet tissue.

Case 7: 40-year-old white male—54–226 (J.L.).

The deceased was picked up by police on charges of drunken and reckless driving and taken to the Parish Prison at 1:00 a. m. He was in apparent good health until 9:00 a. m. of the same morning, when he suddenly developed a generalized convulsion and frothing at the mouth. When seen by a prison extern, he was spastic and rigid, with a frothy material exuding from his mouth. The lungs were clear to auscultation and the heart rate was normal. He became cyanotic about the face and upper thorax, respirations became rapid and shallow and heart sounds distant. Intracardiac adrenalin was administered, with no avail, and he was pronounced dead by a Charity Hospital physician at 10:00 a. m.

Autopsy: The body was that of a well-developed, well-nourished, white male weighing 184 lbs. There were no external signs of trauma. The heart weighed 400 grams. The pulmonary conus and artery were distended by fluid blood. The myocardium, valves and coronary arteries were not remarkable. The lungs were soft and air containing and showed no consolidation. There was a small amount of whitish mucus in the bronchi. The liver was homogeneous, yellow in color and enlarged. No evidence of fibrosis. The stomach contained a small amount of dark brown material. The small and large intestines were not remarkable. The pancreas, spleen and adrenals were not remarkable. The kidneys were of average size and a dark brown color. There was no gross evidence of injury to the scalp or calvarium. The meninges were normal. Multiple sections of the brain showed no gross lesions. The spinal fluid alcohol was 0.03%.

Microscopic: The findings were similar to the other cases.

Discussion

In seven of ten cases of fatty liver with pulmonary fat embolism, the number and magnitude of pulmonary emboli was as great or greater than that seen in sudden death attributed to pulmonary fat emboli following fractures of long bones (8), and was considered sufficient to have accounted for the death.

When individuals in this series were observed to die (Case 7), the clinical events were sudden and were characterized by a prodromal period of disorientation, confusion; followed by convulsions, cyanosis and death. The clinical impression was coronary occlusion in the majority of cases. In many ways the symptoms resemble those seen in massive pulmonary embolism, secondary to thrombi elsewhere (Cases 5 and 7). In Case 7 the post-mortem observation of distension of the

pulmonary conus and artery was indicative of obstruction to flow of blood in the pulmonary vascular bed and right-sided heart failure. The work of Smith and Hara (7) substantiates the thought that death due to pulmonary embolism, even when only small segments of the lung are occluded by

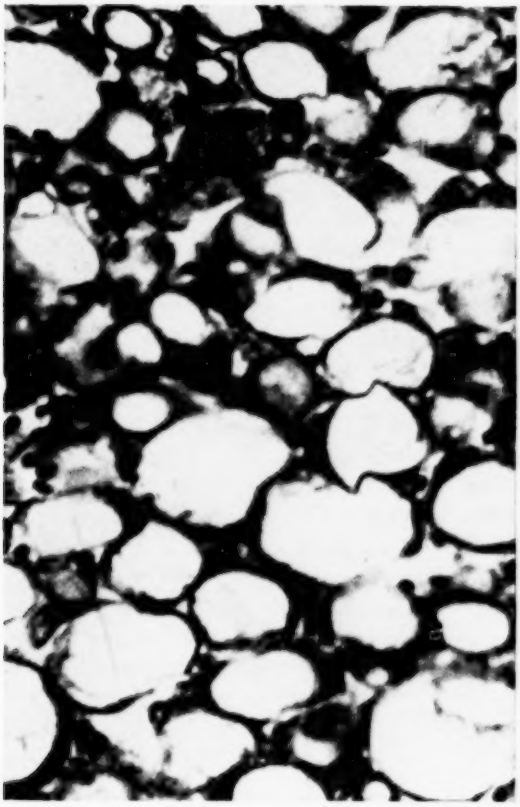

Fig. 1—Section of liver (Case 7) illustrating the diffuse fatty change occurring in the livers of cases of fatal fat embolism. Hematoxylin and eosin. 325x.

emboli, is due to obstruction of the pulmonary vascular bed, spasm or both. It appears that such a mechanism pertains in these cases of pulmonary fat embolism.

Large cysts containing fat (Hartroft's extracellular lipohepatosis) were prominent features in the livers of those cases dead from pulmonary fat embolism. The liver architecture was distorted by these large cysts but there was no significant fibrosis. Many of the cysts in these cases ap-

Fig. 2—Figure 1 at higher magnification revealing the large extracellular fatty cysts with tenuous walls adjacent to sinusoids. The peripheral arrangement of multiple nuclei is evident. Hematoxylin and eosin. 1100x.

peared partially empty and had only a crescent-shaped rim of sudanophilic material remaining inside the cyst wall. In the livers of those cases without pulmonary fat emboli, cysts were less conspicuous and the fat was confined to globules within

the cytoplasm of liver cells (Hartroft's intracellular lipohepatosis). It is probable that the source of the fat emboli is from rupture of these large fatty cysts, with emptying of their contents into the liver sinusoids.

All evidence in this series suggests that the release of fat occurs suddenly and simultaneously from any cysts. The absence of fat emboli in the kidneys, brain and heart indicates that long standing sporadic embolization with subsequent passage of the emboli through the pulmonary capillaries does not occur.

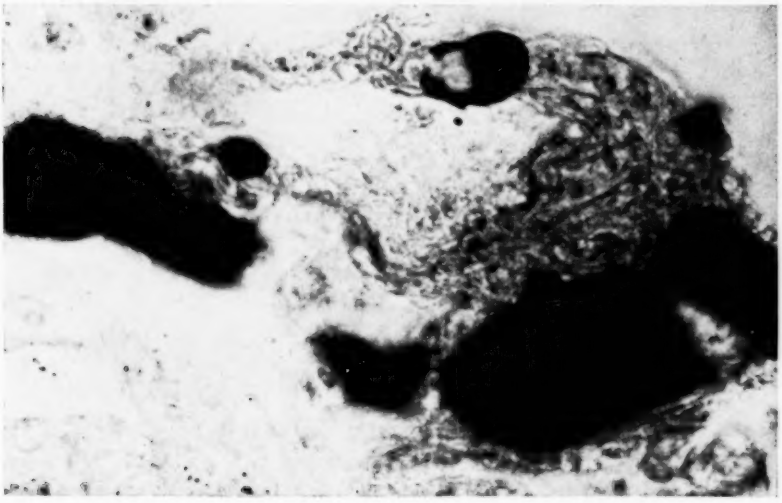

Fig. 3—Section of lung from Case 7 revealing the fat emboli within the lumens of pulmonary arterioles and capillaries. Sudan IV. 900x.

Any possible explanation of the widespread and sudden release of fat from large fatty cysts must encompass sudden changes in intrahepatic pressure. This may be due to slight trauma to the upper abdomen or lower chest over the hepatic area, which in turn produces rupture of the tenuous cyst walls. Sudden changes in intrahepatic pressure due to rapid accumulation of fat or congestion due to alcohol or a yet nebulous vascular mechanism may be responsible. Experiments of Hartroft (4) have shown that rats with a previously

TABLE I
Cases Dying of Pulmonary Fat Embolism

Case No.	Age	Sex	Race	History	Blood Alcohol	Liver * Fat	Lung * Fat	Brain Fat	Kidney Fat
1	67	M	W	Alcoholic found sick in doorway. Death observed in emergency room. No trauma.	0.0	xxx	xxxx	0	0
2	45	M	W	Alcoholic—expired on stoop of home. No trauma	0.22	xx	xxxx	0	
3	61	M	W	Found dead in shower—attending convention. No trauma.	0.30	xx	xxx	0	0
4	60	M	W	Found dead in bed. Alcoholic. No trauma.	0.30	xxx	xxxx	0	0
5	32	M	C	Seen by landlady before expired with dyspnea. No trauma. Known alcoholic arrested for drunkenness.	0.11	xxx	xxx	0	0
6	27	M	C	Found dead in bed in jail. No trauma.	0.0	xxx	xxx		
7	40	M	W	Died suddenly in jail after arrest for drunkenness. No trauma.	0.03	xxx	xxxx		

* Sections cut at 10 mu—unembedded. The degree of emboli is graded according to Robb-Smith (6):

x—1–2 emboli/5 sq. cm.
xx—1–2 emboli/sq. cm.
xxx—1–2 emboli/30 sq. mm.
xxxx—Several emboli/sq. mm.

TABLE II
Cases with Minor Degrees to No Emboli

Case No.	Age	Sex	Race	History	Blood Alcohol	Liver * Fat	Lung * Fat	Brain Fat	Kidney Fat
8	52	M	W	Known alcoholic. Found dead in lot. No trauma.	0.0	xxx	0		
9	25	F	W	Alcoholic—found dead. No trauma.	0.45	xxx	0		
10	49	M	W	Alcoholic—found dead. No trauma.	0.43	xxxx	0		
11	43	F	W	Alcoholic—had been on spree.	0.10	xxxx	0		
12	56	M	C	Found dead in flop house.	0.0	xxx	0	0	0
13	21	F	C	Alcoholic—seen by M.D. two hours before death for weakness and stupor.	0.0	xxx	0		
14	62	M	W	Found dead in jail—arrested for drunkenness. No trauma.	0.14	xxx	0		
15	69	M	W	Found dead in bed.	0.0	xx	x		
16	52	M	W	Found dead.	0.08	xx	x		
17	34	M	W	Died suddenly in jail after arrest for desertion.	0.00	xxx	0		

TABLE II (con't)

Case No.	Age	Sex	Race	History	Blood Alcohol	Liver * Fat	Lung * Fat	Brain Fat	Kidney Fat
18	33	M	W	Died in accident room—vomiting, convulsions and coma.	0.00	xxx	0		
19	24	F	C	Found dead in bed.	0.08	xxx	0		
20	57	M	W	Died in jail. Arrested for drunkenness.	0.00	xxx	0		
21	43	M	W	Collapsed in bar.	0.00	xxxx	0		
22	55	M	C	Found dead in room.	0.15	xxx	0		
23	62	M	W	Found dead in city dump.	0.08	xxx	x		
24	37	F	W	Found dead on floor.	0.12	xxx	0		
25	54	M	W	Died in jail. Arrested for drunkenness.	0.00	xxx	0		

* Sections cut at 10 mu—unembedded. The degree of emboli is graded according to Robb-Smith (6):

x—1–2 emboli/5 sq. cm.
xx—1–2 emboli/sq. cm.
xxx—1–2 emboli/30 sq. mm.
xxxx—Several emboli/sq. mm.

fatty liver treated suddenly and intensely with lipotropic agents in the presence of a high caloric intake may suffer an immediate but transitory increase in liver fat with subsequent release of lipid into the circulation in embolic form. The fact that so many deaths in this series occurred soon after the confinement of the individual to jail suggests the possibility that such a mechanism may operate in some of these cases. Harman and Ragaz (3) have shown an experimental fat embolism in rabbits using intravenous injection of homologous fat, that death by fat embolism is determined not only by the quantity of the fat but also by the precondition of the animal. The mortality in their series was increased by previous dehydration and tourniquet shock. The possibility of the precondition of the chronic alcoholic with pulmonary fat embolism and its effect on mortality must certainly be considered, although any exact correlation is not possible at this time.

In this study, cases with a history or evidence of trauma have been eliminated. This, of course, does not preclude the possibility that minor trauma to subcutaneous fat may have occurred. It was, however, not considered to have been significant in the cases reported here.

In ten cases of sudden death without evidence of trauma or fatty liver, serving as controls, there was no evidence of pulmonary fat emboli on multiple (5) sections of lung studied with Sudan IV method.

Twenty cases of portal cirrhosis studied by identical techniques showed no pulmonary, cerebral, or renal fat emboli.

Summary

A cause of sudden death in chronic alcoholics with fatty livers is presented with case reports and comments. The degree of fat embolism seen in seven of eleven cases with emboli was as great or greater than that seen in fat embolism secondary to trauma. These seven occurred in a total of twenty-five cases studied. We believe massive fat embolism may be the mechanism of death in about one-fourth of the sudden deaths encountered in chronic alcoholics with severe fatty livers.

The gross and microscopic hepatic findings are presented and a distinction made between the findings in those cases

dead from emboli and those cases with minor degrees of embolism.

The possible causes of liberation of fat from the liver are discussed.

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Foreign Crime Detection Laboratories

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The purpose of this discourse or commentary is to inform the reader very generally concerning the functions and maintenance of several of the leading foreign crime detection laboratories which the writer was privileged to visit during the years 1953 through 1956.

Laboratory equipment and qualifications of experts were considered, as well as questions concerning specialization by experts into various fields. Attitudes, technical conceptions and abstractions, unusual or peculiar qualities, exclusive and uncommon thinking, as well as technical practices and procedures were carefully studied. The usual methods and principles employed by technicians in America were used as a yardstick in measuring and studying the above. In general, there appeared to be no extraordinary or unusual deviations, with the exception of a few on which the writer will comment.

Scotland Yard

Scotland Yard, located in London, England, was one of the first organizations visited. On November 18, 1953, a complete tour was made of its laboratory. It has one of seven technical laboratories of the United Kingdom, the other six being strategically located throughout the realm. The laboratory is divided into three sections; namely, (1) physiological, (2) biological, and (3) chemical. It handles nearly all phases of

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technical examinations which fall within the three sections mentioned and includes the handling and processing of latent fingerprint cases. The writer was impressed with the fact that this was the home of the "Father of Fingerprint Classification," Sir E. R. Henry. The laboratory has on display many items of his work and some equipment he used. It also handles some of the less important document cases; however, those of greater import are referred to Dr. Harrison, the outstanding handwriting expert of the United Kingdom, who is located at Cardiff, Wales. In addition, the laboratory has a so-called "Black Chamber" which houses many interesting and famous cases which the Yard has solved over many years.

The equipment used in the laboratory at Scotland Yard is rather out-dated, this being particularly true of the equipment used for microscopical work. Its photographic equipment is adequate, but not of recent date. The laboratory, like so many others the writer visited, suffers from lack of operating funds. Various scientists who are employed as lecturers at some of the leading universities, are called upon to aid and assist, or take charge of a particular phase of an important investigation.

The Scotland Yard laboratory employs thirteen technicians, five of whom are qualified to testify regarding various types of laboratory examinations. Eight of the thirteen employees are apprentices. The technicians take a cautious attitude regarding scientific findings and keep within certain well-defined limitations. For example, the Yard requires 18 characteristic points for a positive latent fingerprint identification which, in the opinion of the undersigned, is ultra-conservative.

Forensic Science Laboratory, Preston, England

On December 14, 1953, the writer visited the Forensic Science Laboratory at Preston, England. It is under the direction of Dr. James B. Firth and handles the following types of cases: narcotics, blood test for alcohol, blood and urine examinations; toxicological examinations, and those involving general chemical analysis. Examination of seminal stains, woods, hair and fibers, and matters pertaining to bacteriology, such as food poisoning cases, etc., are handled

here as well. This laboratory is equipped to examine various textile fibers. For the more difficult textile examinations, it utilizes the research laboratories of several nearby textile companies. Technicians and consultants of local universities and special experts are available on a retainer basis. The laboratory also handles cases involving moldings and casts, such as footprints and tire tracks. However, it does not possess a reference collection of footwear or tires for comparison. Only a direct comparison is conducted when a suspected item or article is submitted. This laboratory is equipped and staffed to handle fingerprint, handwriting, and petrological examinations. It also employs a part time pathologist. Cases involving instrument and tool markings, paint examinations, examination of fingernail scrapings, glass fractures, and firearms identification are also handled.

The Preston Laboratory was well equipped and staffed with competent experts. The writer was particularly impressed with the excellent photographic results obtained by its photo technicians. Members of this laboratory were very interested in techniques employed by American experts. This laboratory is under the direction of the Home Office of Great Britain and supplies laboratory services to police agencies covering the northwest region of England, an area inhabited by 15 million people.

Lancashire Constabulary Laboratory, England

The Lancashire Constabulary, located near Preston, is similar to a state patrol in America. It is manned by more than 3,700 employees, is the largest of its kind in England, and handles criminal as well as highway patrol problems. It is equipped with an excellent fingerprint and photographic laboratory. This laboratory is separate and distinct from the forensic science laboratory at Preston. It is well equipped and competently staffed to handle any latent fingerprint and photographic work. The members of this organization, including the laboratory technicians, are all specially selected and are required to meet rigid physical, mental, and moral standards. They function in a military fashion under the direction of a former military officer.

Prefecture of Police Laboratory, Paris, France

On November 14, 1953, the identification bureau of the Prefecture of Police, Paris, France, was visited. This laboratory handles various types of evidence involving chemicals, metallurgy, firearms, and latent fingerprints. It is equipped with a photographic laboratory. The equipment throughout the entire laboratory was adequate, although not of the modern type. Here a distinguished identification expert, Alphonse Bertillon, worked. The French people are very proud of the great Bertillon and the technical advancements which he had made in the identification field. They have his chair and famous measuring instruments carefully preserved for posterity. The writer was fortunate to be able to examine and study the instruments and other items which Monsieur Bertillon had invented and perfected. Only in this laboratory were technicians found to wear long black smocks, while in other countries either white smocks or none at all were worn in the laboratories. This laboratory does not have a Questioned Document Section for the reason that it is claimed handwriting is not a science and, therefore, is not too heavily relied upon. It was observed by the writer that most foreign countries, particularly those on the European continent, take a rather dim view of the scientific principles as developed in this country regarding handwriting identification and appear to stress the graphological aspects thereof. These aspects are rather delicate subjects in discussions with foreign technicians concerning document identification. The writer is not here attempting to belittle or depreciate the ideas and conceptions of certain foreign technicians in regard to handwriting identification aspects involved in the questioned document field. These observations are made purely as a matter of interest and information. The evidence rules in this laboratory were somewhat lax with respect to the "chain of evidence." Inquiry was made regarding this, and it was explained that the word of the technician in the French courts was freely accepted without any question with regard to the identification of evidence.

Federal German Police Laboratory, Wiesbaden, Germany

The laboratory at Wiesbaden was visited on several occasions between October 1953 and September 1956. It handles the following types of laboratory examinations: toxicological; metallurgical; biological; chemical; spectroscopical; petrological; firearms; tool and instrument marks; wood identification; shoe and rubber footwear identification; narcotics; paint identification; clothing identification (textile weave); flaws and defects in materials; arson; hairs and fibers; powders and explosives; mechanical impressions; fingerprints and questioned documents. A detailed report was prepared by the writer on all examinational matters covered by the laboratory. For the purpose of this paper a report regarding only document examination will be included. The following is a detailed listing of the functions and scope of the document section:

a. Proof of falsification
   (1) Documents and/or partial falsification of security, stocks, and other comparable documents.
   (2) Documents of erased and/or obliterated writings and impressions.
   (3) Deciphering of charred documents.
   (4) Restoration of latent writings and impressions.
   (5) Examinations of inks, stamp pad ink, and writing material.
   (6) Determination of age of inks.
   (7) Paper examinations.
   (8) Glue examinations.
   (9) Examinations of illegal opening of letters.
b. Handwriting examinations
   (1) Comparison and identification of handwritings.
   (2) Psychodiagnostic judgment of handwritings.
   (3) Service for analyzing of handwritings—Central Collection of Handwritings.
c. Typewriting and type-face examinations
   (1) Determination of kind of typewriter used.
   (2) Identification of typewriting.

(3) Examination and determination of type-faces.
(4) Central service for examining of typewriting.

d. Collection for comparative purposes
This collection contains writing materials of all kinds, such as pens, pencils, copying pencils, inks, typewriter ribbons, carbon papers, glues, erasures, stamp pad inks, India ink, writings of various kinds, printing and wrapping paper, and miscellaneous papers.

This laboratory has installed and operates a forgery file used for the comparison of handwritings when a suspected master forger or ring of forgers is operating on a Europe-wide scale. It is similar to the National Fraudulent Check File operated by the FBI in the United States. Of all the foreign crime detection laboratories visited, this one is the most outstanding. It is housed in a convenient but secluded spot on the outskirts of Wiesbaden, Germany. The entire laboratory is very neat and orderly and operated in a highly professional manner. Its technicians wear spotlessly white smocks. It is staffed with 60 technicians, 18 of whom are "Master Experts" and 42 apprentices. The latter must serve a very rigid apprenticeship over a period of several years. They are diligent, precise, and persevering workers. The Experts are highly specialized in their own field. Evidence is immediately receipted, cataloged, and placed under lock and key.

Here the equipment is the latest and most modern. Some has been specially designed. The Questioned Document Section is manned by six Experts, headed by the famous Herr R. Mally.

Any Americans interested in visiting European crime detection laboratories are urged to place the Federal German Crime Detection Laboratory at Wiesbaden, Germany, high on the list.

Federal Technical Crime Detection Police Laboratory, Madrid, Spain

On November 8, 1955, a visit was made to the above named laboratory, which is headed by Senor Florentino Santamaria Beltran. Senor Santamaria has been connected with the

Spanish Crime Detection Laboratory for many years and is a Questioned Document expert in his own right. In the Fingerprint Identification Branch, Senor Santamaria and his associates explained in some detail how fingerprints are classified, how filed, and the system of indexing, cross-indexing, etc. Deceased individuals, as well as new-born infants are fingerprinted for identification purposes. Footprinting of infants is practiced by all leading hospitals in Spain. In this Branch, the Vucetich Fingerprint Classification system is used, and is the same system used by the French Identification Laboratory in Paris. A Latent Fingerprint Section manned by well qualified fingerprint technicians handles latent fingerprints, palm prints, and footprints from crime scenes. Single fingerprints of known criminals, as well as latent fingerprints are classified under a simple but effective system. A rather unique and effective "Moniker" file is in operation in the laboratory. It is broken down to cover age, weight, height, and peculiarities of the individual; however, the primary breakdown is by age.

A photographic section is adequately equipped but is not modern. The Spanish Experts have keen ingenuity in developing and building various types and kinds of equipment for their use, principally because of lack of sufficient operating funds.

The chemical laboratory was being rebuilt; therefore, the writer was unable to evaluate its functions.

The Federal laboratory as a whole was very orderly, neat and clean. It is staffed with well qualified technicians, many of whom have been in a particular specialty for many years.

Several interesting sidelights were noted. An Expert in this laboratory must complete certain technical studies. After completion thereof he must then serve an apprenticeship and further his studies by supplemental readings. Senor Santamaria explained that a technician having once been approved by a court of law as an Expert is henceforth considered as an expert in that particular field. While Spain was a Republic it had a jury system; however, it was abolished during the present regime. At present a court is comprised of the President-Judge and two magistrates who must have had years of previous training in order to qualify. A defense attorney is

not permitted to "brow-beat" or even question the integrity and honesty of a qualified Expert. He is for all practical intents and purposes an Expert of the Court. He merely presents his testimony and demonstrates the reason for his findings. The defense attorney may secure his own Expert and present him as a defense witness. However, this is done only in cases in which there is a doubtful or no conclusion opinion presented by the State Expert. Senor Santamaria took great pride in the fact that the integrity and honesty of his qualified Experts is never doubted. Spain has only this one crime detection laboratory under the jurisdiction of the Federal government. No other official laboratories are sanctioned. Evidence involving all types of criminal cases in Spain is submitted to this laboratory for examination and report. Strict rules regarding the custody and chain of evidence are carefully adhered to.

This Federal laboratory also is the headquarters for "interpol" meaning "international police." "Interpol" handles all matters concerning identification problems on an international scale, such as counterfeiting, swindlers, criminals, etc.

No technician is more sincere than Senor Santamaria. This sincerity was reflected in all of his associates. During this visit numerous cases of unusual interest were discussed and were well supplemented with photographs, photomicrographs, and photomacrographs of excellent quality. Senor Santamaria enjoys an international reputation as a technician in the identification field. He has appeared before the General Assembly of the International Criminal Police Commission Conference held in Oslo, Norway, in June 1953 and at a more recent meeting of this Commission in Turkey. He has delivered papers regarding some recent techniques developed by him, including a new method of evaluating fingerprint and palm print ridge characteristics.

Summary

Our foreign colleagues in the questioned document field are very alert to current and future problems and are greatly interested in progress made by American experts. Several are attempting to delve deeper into the research field to not

only test old and develop new identification principles, but to design and develop new instruments and equipment. Quite a bit of activity was noted along these lines in the German and Spanish laboratories.

With the above in mind, it is respectfully suggested that those of us who are so inclined, be encouraged to correspond more frequently with our foreign colleagues in these matters. The exchange of ideas is the fountainhead of knowledge and progress. It is certain that we are all aware of the fact that progress in any technical field can only be made through and by the experiences of others. The writer hopes that this will be the beginning of a great surge towards better understanding among experts and to the betterment of the profession as a whole.

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History of the International Academy of Legal Medicine and Social Medicine

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For a good many years there existed in a large number of European and South American countries, national societies of legal medicine which later on also had for objectives social medicine, industrial medicine, and insurance medicine.

These societies each year organized international congresses in French, German, Italian, etc., where problems were discussed in forensic medicine, legal psychiatry, criminalistics, toxicology, hazards of accidents at work, and social medicine. Between these disciplines close contacts existed, because they all concur in establishing the relationships which are present between man and legal and social rules to which societies have voluntarily submitted themselves. Therefore, it appeared necessary to fuse into an international association these different disciplines so close to one another.

Thus it was in September 1938 that the International Academy of Legal Medicine and Social Medicine was founded at Bonn, where for the first time the International Congress of Legal Medicine convened.

Although it includes within its activities a large number of disciplines, our association saw its first title abbreviated into "International Academy of Legal Medicine and Social Medicine."

The late Professor Leclercq, the prominent medicolegal French physician, presided at the foundation of this Academy.

* General Secretary, International Academy of Legal Medicine and Social Medicine.

It is to Professor Knud Sand, our distinguished colleague, incumbent of the Chair of Legal Medicine at the University of Copenhagen, that befell the honor of presiding at the first steps of this Academy. Alas, the noise of the weapons which could already be heard in September 1938 was to give rise less than a year later to the explosion which was to tear the entire world for five long years. This cataclysm, without precedent in history, was to suspend the activity of our Academy.

But as soon as the war was over, at the Congress of Legal Medicine held in the French language in Paris in 1946, Professor Knud Sand, with our colleagues Piedelievre from Paris, and De Laet from Brussels, prepared the bases for restoring the activities of the Academy. Finally, on June 25, 1947 the Second Congress of the International Academy of Legal Medicine and Social Medicine was opened in Brussels. During this post-war and rather difficult period this Congress held its meetings in Brussels and in Liege, gathering more than 200 members representing 19 countries from Europe, the Americas, Asia and Africa. More than one-hundred communications and reports were presented. The official organ of the Academy—called "Acta Medicinae Legalis et Socialis"—was finally launched. The former publication was "Zacchia"; the war interrupted its publication.

Soon the Academy was recognized as a member of the C. I. O. M. S., and became sponsored by the UNESCO and the O. M. S. With this help, the publications and later meetings of our Academy were made possible, and underwent new developments.

In spite of the impossibility to organize for purely material reasons the Congress which was planned in Cairo (?) in 1950 organized the Praesidium of the Academy, then composed of

Professor Knud Sand, of Copenhagen
Professor De Laet, of Brussels
Professor Gray, of Toronto
Professor Mazel, of Lyon and
Professor Teisinger, of Prague,

and decided to hold on June 3–4, 1952 a plenary conference of the International Academy of Legal Medicine and Social Medicine, which included 31 countries. Finally in 1955 the

Fourth International Congress of the Academy was held in Genoa under the presidency of Professor Maccaggi. Thirty-eight countries were present; we are going to mention them because it shows how much the Academy was capable of assembling representatives from the entire world. At the magnificent Congress of Genoa, with 300 participants assembled, the following were present:

> Argentina, Austria, Belgium, Brazil, Canada, Colombia, Denmark, Egypt, Ireland, Spain, United States, Finland, France, Greece, Great Britain, India, Iraq, Iran, Iceland, Italy, Japan, Luxemburg, Malta, Mexico, Norway, The Netherlands, Peru, Poland, Portugal, The German Federal Republic, Roumania, Sweden, Switzerland, Syria, Czechoslovakia, Turkey and Yugoslavia.

During this last Congress all the disciplines of legal medicine, industrial medicine, social medicine, psychiatry, toxicology, criminalistics merged in a remarkable harmony.

The interest that so many physicians, chemists, research workers showed for the International Academy of Legal Medicine and Social Medicine demonstrated that the latter is the answer to a need for coordination to which we hope they will continue to give an even greater impulse.

It is with great pleasure that we have seen an American Academy of Forensic Sciences being founded, with which our great desire is to collaborate more and more each day in order that the bonds which unite the International Academy of Legal Medicine and Social Medicine and the American Academy of Forensic Sciences shall become more intimate.

The Praesidium of the Academy includes:

Honorary Presidents: Professors Jules Leclercq * (Lille)
Knud Sand (Copenhagen)
Maurice De Laet (Brussels)
Rene Piedelievre (Paris)

Presidential Committee:
President: Professor D. Maccaggi, Via de Toni, 12, Genoa
Vice-Presidents: Professor M. Ishikawa (Tokyo)
Professor C. E. Paz Soldan (Lima)
Professor E. Wolff (Stockholm)

* Deceased.

INTERNATIONAL ACADEMY OF LEGAL MEDICINE

General Secretary: Professor G. Canepa, Via de Toni, 12, Genoa
General Secretary in charge of the editing of Acta Medicinae Legalis et Socialis, and
Treasurer: Professor P. Moureau, 1 rue des Bonnes Villes, Liege.

Professor of Legal Medicine
University of Liege
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Translated from the French by
Jean-Louis Riehl, M.D.

CERTIFICATE IN FORENSIC PATHOLOGY

The American Board of Pathology is now considering applicants for certification in the special field of forensic pathology. The Board is also accepting applications from institutions that are seeking approval of programs for training in the special field of forensic pathology. For information, write to the Secretary, Edward B. Smith, M.D., American Board of Pathology, Indiana University Medical Center, 1100 West Michigan Street, Indianapolis 7, Indiana.

Psychiatry and Criminalistics*

Maier I. Tuchler, M.D., Phoenix, Arizona

Criminalistics may be briefly defined as the science of crime detection which was the original meaning of the word as coined by Hans Gross. Criminalistics is based upon the application of mathematics, physics, chemistry and the biological sciences and medicine to a study of physical evidence. It is, fundamentally, a physical science. Much of the success of the student of applied criminalistics depends upon his general fund of scientific knowledge and upon his ability to utilize newer and latent methods to develop objective evidence.

Psychiatry, in contrast to criminalistics, deals mainly with the mental and the emotional state of an individual and for years has been concerned with the determination of the mental condition of an accused with reference to the existence of any mental disease or defect which would affect the individual's judgment, responsibility and action. In fingerprinting, the analysis of blood stains, in technical photography, or in the physical measurements of individuals accused of crime, there is little which psychiatry and criminalistics have in common, save a common purpose, subject and goal. For, as ancillary forensic sciences, both psychiatry and criminalistics seek merely to ascertain fact. Both sciences serve as a protection for the innocent, and each science establishes evidence of criminal behavior as is observed either at the scene of a crime or within the mind of man.

Some areas of criminalistics come closer to psychiatry than others; scientific lie detection, and much of the psychology of testimony, appear to sit somewhere in the middle of psy-

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chiatry and often are untouched by psychiatric procedure; other areas of criminalistics are involved with the activities of individuals who are indeed fit subjects for the psychiatric couch, as for example, the recent collaborative study of poison pen letters by a document examiner and the psychiatrist. And, as all students of criminalistics know, repetition and compulsion are significant as the inner core of neurosis; thus, the student of criminalistics finds the arsonist at the scene of his fire, the checkwriter through his signature, and the confidence man by his conceit and naivete. In so many areas do criminalistics and psychiatry overlap that the student of criminalistics must be well endowed with an aptitude for and an interest in the motivations of his fellow man. For this seems to be the case: some men appear gifted with the cold facts of science and have uncanny abilities to develop latent evidence into demonstrable fact; other men, while competent in the biological and physiochemical aspects of criminology, are remarkably well endowed with intuition, imagination, and a temperate interest in motivation, and seem gifted in the use of the tools of science in the study of behavior. This area of aptitude is uniquely interesting to the psychiatrist for much of the interest in investigative pursuit has its origin in late infancy and earliest childhood and is ofttimes readily demonstrable in pre-adolescence and adolescence. However, we are today not concerned with the biological characteristics of the student of criminalistics, although it may in the future be fruitful to study the character structure and personality of forensic specialists. This, by the way, has been done in certain sections of the psychiatric fraternity with interesting and valuable results.

Broadly speaking, criminalistics appears to be concerned with the deed, while psychiatry with the character of the doer, particularly regarding one aspect of his state of mind. The function of the psychiatrist within the framework of the forensic sciences is relatively clear-cut. It is his task to study the defendant after a formal study of the facts on record, to develop a background and a significant past history, and on the basis of a cautious, careful and detailed clinical examination and investigation, explore and clarify those hidden dynamics which may make comprehensible such behavior which

may be categorized as criminal. The psychiatrist studies a criminal from a psychodynamic point of view, applying that massive amount of data gathered by means of psychoanalytic method to the individual case under investigation, and it is his task to formulate the knowledge gained into a language comprehensible to the jury, the judge, and the attorneys.

Forensic psychiatry is concerned with the individual case, separate and distinct, and is not particularly concerned with the crime as such. The psychiatrist is interested in differentiating whether an individual did an act as the result of a conscious, deliberate and voluntary plan of intention, or whether the act, whatever it may have been, was the result of forces involving that level of emotional life below the level of consciousness. For, if an act is truly detached from consciousness, such an act is out of the control of the volition of the individual accused and herein lies much of the difficulty of understanding between the forensic sciences individually, psychiatry, and the law. It cannot be overemphasized nor sufficiently asserted that the mainspring of those forces, which make man act often unwittingly and unwillingly and against his and society's best interests, lies in the unconsciousness. Within this vast area of the unconscious lies the source for the *motivation* of much of aberrant behavior which constitutes criminal action. The origin of much antisocial behavior lies in an emotional and affective undercurrent of activity which results in conscious and overt behavior. Freud's discovery of a dynamic active unconscious has deeply affected the traditional views on crime and criminology and the influence of present day psychoanalytic psychodynamic theory and practice is being felt in all areas of forensic science. For example, a rookie policeman is identified by an experienced police chief as a part-time burglar and arsonist, the interest in the criminal behavior of an overly enthusiastic rookie police officer is not limited to the formal and procedural aspects of detection, conviction, and disposition, but involves the police chief, the district attorney, the judge and practically all with any contact in the case with an interest and intense curiosity concerning the motivations of the accused. There certainly appears, as case after case had demonstrated, an intense interest, not only in the how of a

criminal act, but also in the why of such act. Here, forensic psychiatry serves a distinct purpose and can make a definitive contribution.

Common to both criminalistics and psychiatry is the necessity to translate, ofttimes, complicated scientific data into reasonable and everyday language, comprehensible to the average jury. The more complicated the scientific field, the more incomprehensible at times the clarification, and the more vulnerable the specialist becomes in cross-examination. While the presentation of data to the court involves a skill in communication, which often overshadows the technical knowledge of the forensic scientist, the problem of presentation of data is often extremely difficult to the forensic psychiatrist. The problem of psychiatric presentation of data appears rooted in the fact that there are indeed three points of view which, in every case, must be interpolated and expressed in common everyday usage. Perhaps a presentation of a paper on psychiatry and criminalistics before a group of police scientists illustrates the interest latent in related specialties but rarely in communication with each other because of what might best be called "language barriers."

A psychiatrist examining a case formulates the data gathered in a language of psychodynamics not incomprehensible to the intelligent layman but so remote from everyday usage as to demand, for any value to a judge or jury, a simplification, clarification, and, if clinical terms must be used, a re-definition. The late Lowell Selling in every report to the court frequently appended a glossary of those psychiatric terms used and many psychiatric experts incorporate within a report to the court such definitions as are needed for accuracy.

The law, and particularly the criminal law, has a language and a procedure based upon centuries of precedent. The fundamental concepts of our Anglo-American criminal law and the language with which these concepts are expressed constitutes indeed a hurdle which is often discouraging and unrewarding for the forensic psychiatrist. For a forensic psychiatrist is dealing with more than a clarification and simplification of data; there exists a fundamental difference between the legal thought and the psychiatric thought. The

former is conceptual in origin, oftentimes archaic in concept, deeply rooted in precedent, and often so firmly entrenched into usage as to frequently remain unassailable by current psychiatric advances.

The criminal law has little interest in the mental condition of the accused. The capacity to entertain intent appears to be the only condition of mind of significance. If intent concurs in point of time with the criminal act, the act is thus considered the result of intent; an act without intent is an act of innocence; intent without act is only a simulation. The "mens rea" which constitutes a murder means malice aforethought and a "mens rea" in the case of a theft involves an intention to steal. The fundamental requisites for the commission of a crime appear to be related to the capacity of the individual to form an intent; a fundamentally ill person is relieved of responsibility not because of a philanthropic or humanitarian impulse derived of law but because he was considered incapable of formulating intent. This is the basis for a definition of legal insanity. For some centuries, psychiatrists have argued that an insane person who knows the difference between the right and the wrong is capable of forming the intent and, therefore, is to be treated as any other person under the law. This definition of legal insanity, as compared to medical insanity, when based upon current psychiatric knowledge, is fairly meaningless. Incorporated within the McNaughton rules as formulated in 1843, this doctrine has been successfully challenged in the Durham decision of 1954 and within the next decade the determination of the sanity or insanity of an individual convicted of a crime may become a more psychiatric undertaking than a legal formality. The concepts upon which sanity or insanity are based were derived at a time when little was known of the workings of the mind and the concepts of legal insanity, while written in the language of law, were based upon static concepts of early nineteenth century psychology.

Present day forensic psychiatry is involved, in addition to evaluation of the medically and legally insane, with the exploration and evaluation of the non-insane criminal offender.

The psychiatrist embarking on a witness stand without an intimate familiarity with the definitions and concepts of the law is often a babe in the woods, surrounded by experts trained in the explicit meaning of words which don't mean what they say. For example, as Diamond (1) has pointed out: "Malice aforethought is a special type of criminal intent which, at the present time, has nothing to do with ill will, hatred, enmity, or the ordinary meaning of the word 'malice.' Furthermore, there is no implication of premeditation, contrary to the conventional meaning of the word 'aforethought.'" Legal terms have become highly specialized; they have changed their meanings throughout the centuries and they can only be understood when studied in historical sequence. As Diamond (2) has stated: "In this respect the historical study of the law is closely analogous to the analysis of our patients, and symptoms that are seemingly contradictory and incomprehensible become fully meaningful through the genetic approach (historical)."

The distinction between manslaughter and murder involves the presence of malice aforethought in one instance and its absence in the other, and first-degree murder is defined as a homicide committed with malice aforethought and with premeditation. The dilemma of the psychiatrist in forensic practice can become very puzzling when it becomes his task to ascertain the state of mind of an individual at the actual instant of an assault committed many months in the past; and this task becomes almost an impossibility when, after a study of the non-insane criminal, and of the normal human individual active in the work of the world, we find, buried in an unconsciousness, occasionally coming to consciousness as an aberrant thought, frequently persistent as a neurotic symptom, thoughts of "inward intention, as lying in wait, menacings antecedent, former grudges, deliberate compassings, and the like" (3). Hale's definition, quoted above, implied, "malice is a deliberate intention," and herein lies one of the fundamental difficulties in the application of psychiatry to forensic practice: intention appears the criterion of the guilty mind while motivation is disregarded under the law. Further removed from the concept of motivation and

even removed from the concept of malice aforethought are felonies which are designated so even in the absence of premeditation or malice aforethought; for example, an unintentional killing during the commission of a robbery.

Although the law specifically states that motive is irrelevant: "For hardly any rule of penal law is more definitely settled than that motive is irrelevant," (4) and it presumed that within a definition of malice aforethought, one assumes *how* the mind of a criminal functioned at the time of the crime, there still remains that motivation, the why of the act, is the reasonable concern of the psychiatrist and, despite insistence that motive is irrelevant, appears the preoccupation of the lawyer. In the strictest sense, a jury is impaneled to bring in a verdict knowing the how of a crime; the why is irrelevant. Criminalistics establishes the how of a given crime, the why is often established by a chain of circumstantial data which is used to establish a motivation. The hypothetical case is often used to establish motivation. An attorney argues motivation in his argument to a jury for the jury is interested in motivation, as indeed are the attorneys and the judge; so why all the protest which restricts the presentation of evidence or data which might apply motivation deeper than the cold facts on record, data which may make comprehensible some incomprehensible act?

Perhaps here, criminalistics and psychiatry find a common ground; both the psychiatrist and the criminalistic specialist speak as expert witnesses and each should be given the opportunity for the full expression of their findings and of their opinions regarding their findings. The competent expert is not afraid to expose his data to the most severe criticism, the intelligent cross-examination, and he must be able to explain the groundwork upon which his temperate decision and opinion is based. Many a competent investigator in police science has been hampered by limitations of a purely legalistic nature; so, in psychiatry the inability to present the concept of the motive to its fullest may seriously handicap a defense, and often a prosecution, and it is becoming increasingly apparent to the forensic expert that he be legally permitted to the full clarification of his data. In psychiatry this refers to the opportunity to present a comprehensible motivation in

the place of an often incomprehensible and extremely nebulous intent.

Summary

While criminalistics and psychiatry are often far removed in techniques, the subject matter involves man, his motives, his intentions, and his acts. Both the forensic psychiatrist and the specialist in crime detection follow the scientific method and both study the genetic or historical development which led to consequences of an act, and elaborate on that evidence and data presented during the consummation of an act, equally to protect the innocent and/or to explain the guilt of the accused.

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Recent Progress in Forensic Pathology*

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Introduction

The practice of forensic medicine is usually defined as the application of medical knowledge to the needs of the administration of justice. With the increasing propensity toward litigation which is so much a part of our current scene, there is scarcely a disease or disorder, congenital or acquired, natural or traumatic, whether it be degenerative, infectious, neoplastic or metabolic, which has not moved from its usual native locus in the clinic, operating amphitheater, hospital bed or autopsy room and made its appearance before some tribunal. In adjudicating these issues, the testimony of a well-trained, conscientious physician is essential if a just verdict is to be reached. No specialty in medicine confers immunity from a subpoena. Industrial disease and accident, within the meaning given by many of the workmen's compensation codes, cover practically every entity listed in the Standard Classified Nomenclature of Disease. Inasmuch as morbid anatomy is the organic basis and substrate of clinical symptomatology and signs (and we should include morbid mental states as the foundation of functional complaints), forensic pathology enters into, embraces and bottoms the entire field of forensic medicine . . . really medicine in its broadest sense. Even when forensic pathology is limited to

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mean traumatic pathology, its ramifications extend far beyond the immediate traumatic lesion. For example, the relationship of physical injury to the onset of diabetes mellitus or the possible or probable etiologic importance of mechanical trauma to carcinogenesis (1) are medical problems which present themselves in court where the pros and cons are debated in the open arena.

It is fair to say that much of forensic medicine and forensic pathology is rightfully the domain of the clinician. For us here, forensic pathology may be defined in its more narrow meaning as including those phases of laboratory medicine which fall more or less completely within the purview of the morbid anatomist whose primary interest is the acquisition of accurate data and knowledge that bear on the cause, manner, and mechanisms of death and any related phenomena. The clinical or bedside implications of these facts are either non-existent or secondary. This is at best a rather lame and arbitrary definition, but it is useful in delimiting the boundaries of the field.

An attempt has been made to scan the literature published since the turn of the decade and to present a few of the more significant contributions to the field of forensic pathology. Because intellect and presentation time granted to the author are both sharply limited, this survey cannot hope to be complete, and it is probable that important publications have been overlooked or omitted.

Asphyxial Deaths

Shapiro, in a paper entitled Medicolegal Mythology (2), calls for a critical re-examination of the "many tests held dear by the uninquiring mind for many generations." Due to what he terms a "peremptory adhesion unto authority," a large number of vulgar errors and arbitrary assertions have been repeated in edition after edition of textbook after textbook, always on the basis of inadequate authority and unthinking observation.

Nowhere is this situation more true than in asphyxial deaths, or more specifically, deaths resulting from mechanical interference with respiration. Many standard texts imply that an asphyxial mode of death can be inferred solely from

general internal anatomic evidence (3). Sufficient reliable data have been accumulated to demonstrate conclusively that the general anatomic changes frequently attributed to mechanical asphyxia occur in a variety of other conditions including sudden natural death. Gordon and Turner (4) emphasize that such nonspecific findings as visceral congestion, cyanosis, cardiac dilatation and persistent post-mortem fluidity of the blood are not singly or in combination pathognomonic of death by asphyxia.

Cyanosis is a normal finding at autopsy in the absence of conditions that alter hemoglobin, and liquidity of the blood is neither characteristic of an asphyxial mode of death nor a reliable basis from which strong inference can be drawn (3). Acute pulmonary emphysema occurs in relatively few forms of asphyxia, e. g., drowning and the impaction of a foreign body in the respiratory tract (5).

Tardieu's spots, to which great diagnostic significance is occasionally granted, are not indicative of suffocation or asphyxia. They have been noted to occur *after* death from many causes (6), and are completely unrelated to an asphyxial mode of production in the living. Their number, size and distribution are of no importance except insofar as they are found above the level of a compressive mechanism, ligature or otherwise. Rusakoff attributes them to increased capillary permeability with superimposed mechanical factors (5). These subserosal thoracic visceral hemorrhages result in part from suction in the lungs and from cardiac motion. Recent studies place visceral petechiae, in the absence of organic disease attended by hemorrhagic manifestations, in their proper insignificant place (3).

Reports in the literature on splenic anemia stemming from asphyxia are based on animal experimentation. Many animals have in their spleens comparatively strong musculature which contracts with appropriate stimuli and results in partial emptying of the splenic sinusoids. Man does not possess such musculature, and this finding is not applicable to Homo sapiens (5).

Thus, the so-called general signs of asphyxia cannot be credited with the diagnostic importance ascribed to them. They are nothing more than the signs of sudden death, what-

ever the cause. In those dead of asphyxia, emphasis should be placed on local as opposed to general signs, i. e., fingernail abrasions on the skin and injuries to the deeper tissues in throttling and evidence of a constricting ligature in garroting. A diagnosis of asphyxia made exclusively on internal appearances may place the pathologist in a difficult position. His concern should always guide him to sound inferences which can be drawn from scientifically acceptable data. There is no justification for perpetuating the view that asphyxia is a pathologic entity recognizable at the autopsy table.

The attention of the pathologist must thus be focussed on local as opposed to general anatomic evidence of asphyxia. In this connection Prinsloo and Gordon (7) call attention to hemorrhages occasionally produced in the neck during dissection which may be confused with ante-mortem bruises inflicted shortly before death. Even microscopic examination may not differentiate between the two. A suitable dissection technique is described which eliminates these troublesome and confusing artefacts.

Drowning

Attempts to establish the mechanism or mechanisms of death in drowning, and the necessity of finding some means to differentiate death by drowning from death due to other causes with subsequent submersion have concerned many investigators.

The notion that death by drowning is a purely anoxic asphyxial mode of exitus is now regarded as an oversimplification. During drowning aspiration and trans-alveolar absorption of large quantities of water occur (8), and the physiologic mechanisms of drowning may depend on whether the absorbed water is fresh or salt. Animal experimentation and post-mortem chemical analyses indicate that death in fresh water drowning is frequently the result of ventricular fibrillation. The fatal arrhythmia is triggered by flooding of the blood stream with water (9, 10). All the blood constituents are diluted except the serum potassium which rises rapidly to figures as high as 8 m.e.q./L. as a result of hemolysis due to hypotonicity of the plasma (8, 9). Ventricular fibrillation does not occur in salt water drowning (9).

Bowden (11) hypothesizes that sudden elevation of the serum magnesium in salt water drowning may lead to death on the basis of cardiac standstill from electrolyte imbalance. In fresh and salt water drowning fulminating pulmonary edema occurs, accompanied by diffusion of plasma proteins into the alveoli (8), accounting for the frothy, blood-tinged exudate present in the nostrils and mouths of many drowning victims (12). These studies may have important therapeutic applications. Although the immediate resuscitation of the near-drowned remains identical in fresh water and salt water drowning, namely the establishment of a patent airway and the use of artificial respiration, after-care may depend on the character of the drowning medium, e. g., electric defibrillation or stimulation, cardiac massage or parenteral therapy with appropriate solutions.

Because gross anatomic examination of those apparently drowned may fail to disclose the signs usually associated with this type of death, chemical and physical laboratory tests and histologic techniques are being increasingly utilized to establish the diagnosis of death by drowning. The classic Gettler test, limited to analysis of the chloride levels of the right and left heart's blood, does not furnish the desired information in many cases. Accordingly, attention has focussed on studies of other blood constituents and properties, particularly on the specific gravities of the blood in the major and minor circulations. In all drowned victims, irrespective of the salinity of the water, the specific gravity of the left atrial plasma is less than that of the right atrial plasma, whereas in non-drowning controls, the reverse is true (13).

A histologic approach to the diagnosis of drowning is employed extensively by European investigators. During drowning particulate water-borne substances, diatoms, find their way into the air passages and peripheral lung tissue and are disseminated throughout the body by the blood stream following their absorption from the alveoli. Structures up to 30 micra can penetrate readily into the pulmonary circulation during drowning and arrive shortly thereafter in the liver, kidneys and brain. They have been demonstrated in the viscera of experimental animals, even though the animal

has been immersed only a short time and has been removed alive and gasping (14).

The presence of these particles solely in the lung is not as reliable a diagnostic criterion as their being found elsewhere. They are known to reach the subpleural spaces post-mortem, particularly after prolonged submersion. Working with rabbits, Holden and Crosfill (15) concluded that the presence of foreign bodies in the lung does not permit any firm opinion to be expressed as to whether or not life was present when the body was immersed, unless there is widespread dissemination in both lung fields. Small numbers of diatoms are found in the lungs of rabbits submerged after death in heavily contaminated water.

In victims of drowning Naeve (16) demonstrated the shells of diatoms in the sediment produced by digesting 300–400 grams of liver with acid. This finding was absent in "atypical" drowning in very cold water where severe shock supervened rapidly on immersion. Diatoms have been observed also in the bone marrow of the recently drowned (17, 18), and darkfield illumination of "optically empty" tissue sections, especially the kidney, may demonstrate diatoms inhaled *intra vitam* (19).

The number and size of the diatoms in the viscera vary with their content in the inhaled water (20). Hence it is advisable to examine a sample of the drowning medium. All the above findings may be absent if the water is low in diatom content.

An unusual aspect of drowning is reported by Lentino (21) who investigated a body submerged in fresh water for 6 years. He reports a peculiar association of excellent preservation of the gross structure of the thoracic and abdominal viscera with complete disintegration of their histologic pattern.

Inhibition

Sudden and unexpected death where gross and microscopic anatomic findings are negative, and where microbiologic and chemical studies are noncontributory make up a small but important fraction of sudden deaths in healthy persons. Such

deaths have been termed "instantaneous physiologic deaths" (22) or deaths from inhibition (23). The essential factor is apparently the establishment of an inhibitory reflex with sudden arrest of a vital organ or function following microtrauma which is not lethal in the ordinary sense (24). Palmieri (25) hypothesizes that death from inhibition is caused by a vasomotor reflex which leads to accumulation of blood in the splanchnic capillaries. Respiratory and circulatory activity suffer from the resultant decrease in circulating blood volume, a situation aggravated by transudation of plasma through the capillary endothelium.

Simpson (26) calls attention to an important forensic aspect of inhibition by pointing out that a rape victim may die suddenly in consequence of the grip of the hand on the throat, a stimulus which may lead to carotid sinus hyperactivity and cardiac stoppage, a form of inhibition. In the best interest of fair and real justice, he feels that the court should be informed how unexpected and unintentional death may be in such circumstances.

Inhibition or inhibitory mechanisms may play a part in some suicidal or accidental hangings. In most such instances fracture-dislocation of the cervical vertebrae and transection of the cord characteristic of judicial execution do not occur. On the basis of pathophysiologic experiments and anatomic findings, Vasiliu and Diaconita (27) conclude that an inhibitory reflex may be rapidly precipitated by sudden stimulation of the subcarotid reflexogenous zone by compression of the rope and weight of the body. This mechanism may explain some cases of fatal hanging in adolescent males who have been playing with no intention of ending their lives (28). On the basis of 7 cases of interrupted hanging, Puccini (29) states that reflex syncope caused by bulbar inhibition due to carotid sinus stimulation is an important part of the hanging syndrome. He interprets the hanging syndrome as resulting from cerebral anoxia due to compression of the great vessels in the neck together with reflex phenomena evoked by stimulation of the carotid sinus receptors, the vagi and the pericarotid sympathetic and parasympathetic nerve plexuses.

Chronologic Phenomena

The forensic pathologist continues to search for reliable criteria and sound bases for establishing the time of death, or better, the range of time during which death might have occurred. He is constantly faced with the problem of fixing the time of death between the limits of probability (30). It is well-recognized that these limits are wider, the longer the interval between the moment of death and the time of examination. As the post-mortem interval lengthens, associated evidence may furnish more reliable data on which to base the time of death than do the anatomic changes.

A related aspect of thanatology is the estimation of the survival interval, the time elapsing between injury and death. These two chronologic facets of death may be the critical issues in homicide investigation, supporting or invalidating alibis.

The classic critera of rigor, livor and algor mortis may leave much to be desired when there is real need for trustworthy data to establish the time of death. Smaller joints are apt to go into rigor more rapidly and earlier than larger joints, and involvement of the entire body by rigor is not always regularly progressive or symmetrical (2). Rigor is thus an indicator of time of death only within wide limits. The assessment of the duration of death from the state of rigor must be made against a background which takes into account all factors influencing post-mortem changes in general, the size and development of the musculature and the type of ante-mortem activity (30).

Cases are described where rigor has appeared and disappeared quite irregularly. A 68 year old man, found in his bed 36 hours after having been murdered, demonstrated rigidity in the uncovered upper half of his body while the covered lower half was flaccid and moderately putrefied (31).

The rate of cooling cannot be used as exactly as the forensic pathologist would desire without taking into account the environmental temperature as well as such other variables as the rate of evaporation. Moreover, the human body does not cool simultaneously and regularly throughout (32).

The rate of progress of a meal along the intestine and the rate of emptying of the stomach may furnish useful data to aid in establishing the time of death, *if* one is familiar with the eating habits and meal times of the deceased, the quantity of the meal and the interval between the last 2 meals. The nature of the meal must also be taken into account. Liquid moves more rapidly that semi-solid, and semi-solid progresses more rapidly than solid. De Saram (30) checked the contents of stomach and intestine of 41 executed men. He placed multiple ligatures along stomach and intestine at distances of 1–2 feet, thus compartmentalizing the intestinal tract. He observed that the stomach starts to empty approximately 10 minutes after the first mouthful, and that it empties sooner with a small meal than a large one. A large solid meal progresses 6–7 feet per hour in the small intestine and reaches the ileo-cecal valve in 3–3½ hours.

Adipocere formation has been intensively studied by Mant (33) who examined several hundred exhumed war dead. Observations in the field were supplemented by controlled laboratory studies. Adipocere arises from hydrolysis of fat, and appears only when fatty tissue is present. It does not form in the emaciated. Fat and water alone are not sufficient to produce adipocere. Putrefactive organisms, of which Cl. Welchii is most active, play an important part. An anaerobic environment thus favors adipocere formation, especially in the presence of moist warmth. Access of air to the body after burial hastens aerobic putrefaction with less adipocere formation. Unburied bodies undergo more rapid decomposition and form less adipocere. Burial in a coffin retards or inhibits the process. Adipocere is most abundant under loosely fitting clothing or body coverings. In mass graves the bodies in the center are less decomposed and show more adipocere than those at the periphery.

Investigation of the mycologic flora to establish even approximately the time of death is reported as essentially without value (34), and no correlation is demonstrable between the degree of hemolysis and the post-mortem interval (35). The microscopic appearance of the epidermis cannot be used to determine the time of death of cadavers found in water (36).

Fundascopic differentiation between real and apparent death has been described. Changes occurring in the fundus after death are said to furnish a means to estimate (within a range of minutes) the time elapsed since death. Clinically these changes may indicate the advisability of attempting resuscitative measures (37).

Estimation of the survival period may be aided by microscopic data reported by Enos et al. (38). Approximately 1 hour must elapse before an appreciable number of granulocytes migrate to an injured site. A heavier infiltrate points to more prolonged survival. In sudden rapid death the spleen contains 10–20 eosinophiles per high power field. The number falls to 1–2 when death occurs ½–5 hours after injury, and after 6 hours there are none. Twenty-four hours after injury the liver cells lose their fluffy quality. Between 5 and 12 hours after injury fluffy *and* opaque liver cells are present.

Needle puncture marks show an orderly progression of changes of value in ageing the lesion. At 4–6 hours the opening is plugged by protein-rich fluid and an early inflammatory exudate. In 24 hours there is a sharply defined disc of leukocytes. Definitive epidermal cells form in 3–4 days, the plug is shed, and the new epidermal cells rise to the level of the surrounding skin in 4–6 days (39).

Still another aspect of traumatic chronology deals with differentiating ante-mortem and post-mortem injuries. Post-mortem injuries, inflicted after molecular tissue death, are readily distinguished from *intra vitam* lesions by the absence of vital reaction. However, vital reaction in the skin is still possible within 10–20 minutes after somatic death. True post-mortem injury of the skin leads to elongation and fascicular arrangement of the epidermal nuclei, a change commonly seen in electrical burns and said to represent a thermal effect after primary necrosis (40).

Small bruises can be produced post-mortem if injury is inflicted within 1–2 hours after death, particularly if the body is still warm. Large bruises are always ante-mortem in origin. Several characteristics of bruises are valueless in helping to decide their ante-mortem or post-mortem inception. Inability to wash out the blood from the bruise, absence of

swelling, abrasion of the skin, and clotting or failure to clot of the extravasated blood furnish no sound bases for differentiation. The changing color of bruises takes days to develop and is of no help in diagnosing bruises inflicted shortly before death from those caused shortly after death. Histologic examination of a bruise may not be contributory in establishing its time of origin because the blood may remain unchanged (41).

Weapon and Wound

Establishing the nature of a missing weapon from the type of injury inflicted is a common problem in the forensic pathologist's autopsy room. Two recent papers discuss this etiologic aspect of traumatic diagnosis. The varieties of wounds produced by scissors are described in detail by Rauschke (42). When the scissors blades are closed, both edges of the wound are covered by introverted epidermis, and the injury may simulate a knife wound. When only one blade penetrates, one edge of the wound is covered by introverted epidermis, and the other edge shows severed elastic fibers.

Matching weapon and wound has been attempted in skull fractures, and experiments have been carried out to discover how closely the size and shape of the fracture corresponds to the size and shape of the responsible instrument. To achieve anything like an *exact* fit between striking surface and fracture, the former must be small, inflicting what is essentially a puncture wound. In the case of hammers and axes, no true imprint in either size or shape is usually present. Depressed fractures can be produced by striking areas measuring up to 20 square centimeters (43).

Soap Abortion

Descriptions of anatomic changes and discussions of mechanisms of death resulting from attempted or successful abortions with soap and soap-like solutions are frequent in European medicolegal writing. This type of death is apparently more commonly encountered in the dissecting room of our European colleagues than in our own. Sudden death in such attempted interruptions of pregnancy is often due to air embolism, but general intoxication due to the soap solution

may lead to severe dyspnea, convulsions and collapse (44). A neurogenic shock syndrome due to stimulation of the autonomic end organs in the blood vessels by the irritant solution has also been proposed as an explanation for sudden death. This mechanism is said to be manifested anatomically by splanchnic vasodilation and acute pulmonary edema (45). The primary changes due to injection of the soap are seen in the internal genitalia where corrosive necrosis of the decidua, chorionic villi and myometrium are most prominent (46). These findings may be masked by secondary infection. General peritonitis may occur from transtubal passage of the soap solution. The necrosis and inflammatory changes result from the free fatty acid radicals, the hydroxyl ions and soap molecules. Hemolysis is striking and leads to hemoglobinemia, hemoglobinuria, hemoglobinuric nephrosis (lower nephron nephrosis), anuria and uremia in those who survive the immediate insult.

No one change is absolute proof of the injection of soap. The development of any or all the abnormalities depends on the nature of the soap solution and the mode of administration. It is thus important to demonstrate as many changes as possible to increase the diagnostic certainty of the events leading up to death.

Summary

A survey has been made of recent contributions to the broad field of forensic pathology. Included are discussions of anatomic findings in asphyxial deaths, the mechanisms and diagnosis of death by drowning, the establishment of time of death and evaluation of the changes arising after death, ageing of injuries, instantaneous physiologic death, matching weapon and wound, and soap abortions.

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TECHNICAL SECTION

Determination of Meperidine in Biological Specimens in Conjunction with a Case of Demerol Intoxication

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Studies of meperidine absorption, fate, and excretion are made difficult by the rapidity with which the drug is metabolized. Several papers have appeared in the literature concerning this subject, but for the most part they deal principally with studies on animal metabolism. A case involving suspected lethal ingestion of Demerol offered this laboratory an opportunity to study the concentrations of meperidine in the various tissues and fluids submitted. For this study a procedure based on the ultraviolet absorption spectra of phenylpiperidine was developed. The phenylpiperidine configuration is responsible for the characteristic meperidine and normeperidine spectra. This procedure is rapid, simple, and affords the high degree of specificity associated with ultraviolet absorption spectra—especially where a recording spectrophotometer is available.

Case History

A thirty-two year old Caucasian male was found unconscious in his home. On hospital examination the upper half of the body was markedly cyanotic,

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and no signs of life could be detected. Artificial respiration, emergency tracheotomy with tracheal aspiration, and intracardiac medication were not effective. The individual was pronounced dead twelve minutes after admission to the hospital. Blood was withdrawn from both femoral veins, and approximately eighty-five milliliters of gastric contents were aspirated. The blood, gastric contents and sections of muscle, liver, lung, and kidney were submitted for toxicological examination.

The gross autopsy description noted that the right forearm showed "an area of discoloration and induration measuring 13.5 cm. in length, and 0.6 cm. in width and starting just above the skin line of the wrist and on the vertical line with the right thumb." The area was marked by approximately eighty punctiform wounds grouped irregularly in clusters and in distinct contrast to the surrounding skin by its dark brown coloration and dry appearance. This evidence and the fact that the deceased had access to Demerol by job association strongly suggested addiction.

Results of the toxicologic examinations are summarized in Table I.

TABLE I

Specimen	Meperidine concentrations (expressed as mg. free base/gm. except where indicated otherwise)
Liver	0.17
Kidney	0.08
Lung	0.34
Muscle	0.07
Blood	0.03 mg./ml.
Urine	1.17 mg./ml.
Stomach aspiration (before autopsy)	0.02 mg./ml.
Stomach content	trace *

* A very high blank and low meperidine concentration made a quantitative estimation difficult. Presence of meperidine was established from results of chromatography (see Table II). This stomach content was different in appearance from that of the stomach aspiration. The dark character of this substance was suggestive rather of the upper bowel or duodenal contents.

Procedure

Urine and homogenized tissue specimens are made alkaline (pH 9.0 or above) with saturated sodium or potassium hydroxide solution and extracted with five parts of ethylene dichloride.[1] The ethylene dichloride should be washed with one-tenth volume of 0.5 N sulfuric acid followed by one or two aqueous washes to remove any traces of the acid. The washed solvent is filtered through Whatman #41 filter paper before use. The five-to-one solvent-specimen ratio is minimal so that a larger volume of ethylene dichloride

[1] Ethyl ether and chloroform are other solvents that were tried, but these consistently produced higher blanks with tissue. The high blanks made a quantitative estimation difficult in some instances.

may be used. In all extractions, shaking for five minutes gave recoveries of 92–94%. Longer periods of shaking—up to fifteen minutes—did not increase recoveries and tended to produce higher blanks. After the layers have settled the entire solvent layer is filtered through Whatman #41 filter paper to remove any of the aqueous phase retained by the solvent. The filtered solvent is washed with M/15 dibasic sodium phosphate (pH 8.5) buffer, and when the layers have separated, it is again filtered through Whatman #41 paper. After filtration, the solvent layer should be clear. Any cloudiness must be removed by refiltration, or high speed centrifugation (3000 r. p. m.) if the volume of solvent is small. As large an aliquot as possible is taken of the clear solvent and extracted with one-tenth (1/10) volume of 0.5 N sulfuric acid. The sulfuric acid extract is then read in the ultraviolet region from 300 to 230 mμ with a spectrophotometer.[2] The recorded spectra of meperidine is shown in Fig. 1. From the four concentrations given in the illustration, the absorbancies at 257–258 mμ can be seen to follow Beer's law.

In Fig. 1, the meperidine spectra begins very sharply at 275 mμ. Any absorbancy at 280 mμ or above can be discounted as interfering substances and dealt with accordingly. For example, if the concentration of meperidine is high, linear absorbancy above 275 mμ may be subtracted from the 257–258 mμ reading (see Fig. 2, A). Another alternative is to add one or two drops (no more) of saturated sodium or potassium hydroxide to the solution in the cuvette and re-read the alkaline solution in the spectrophotometer. This change in pH will not alter the spectra appreciably (see Fig. 1), but it does reduce the absorbancy of some interfering substances (see Fig. 2, B and 2, D). If large amounts of interfering absorbancies persist, a re-extraction of the sulfuric acid extract will eliminate this trouble. Where low concentrations of meperidine are encountered, a re-extraction is almost always necessary.

To an aliquot of the 0.5 N sulfuric acid, saturated sodium or potassium hydroxide is added dropwise only in amount to raise the pH to ten (10) or above as tested with pH Hydrion test paper. Five volumes of ethyl ether are used for the re-extraction, and upon separation of the layers, the ether is filtered through Whatman #41 filter paper to rid it of any aqueous phase. An aliquot of the solvent is then extracted with three (3) or four (4) [3] ml. of 0.5 N sulfuric acid. The acid must be free of ether before it can be read in the spectrophotometer. This is accomplished effectively by transferring the aqueous acid layer to a test tube, and holding the tube under a hot water tap so that the water can circulate along the outside of the tube until the ether odor is no longer detectable. The solution is then cooled, and its ultraviolet spectra determined as before.

For the calculations, two methods are employed to estimate the blank. The first, mentioned above, is to subtract any absorbancy at 280 mμ from the 257 mμ reading. In Fig. 2, A, the meperidine recovered from the urine is indicated

[2] All spectra described in this procedure were obtained on the Cary Model 11 M recording spectrophotometer.

[3] The quantity of acid here is dependent on cuvette size. Usually 3.5 ml. can be recovered from a 4.0 ml. extraction. With larger meperidine concentrations and/or volumes of solvent in excess of fifty milliliters, more 0.5 N sulfuric acid would be desirable.

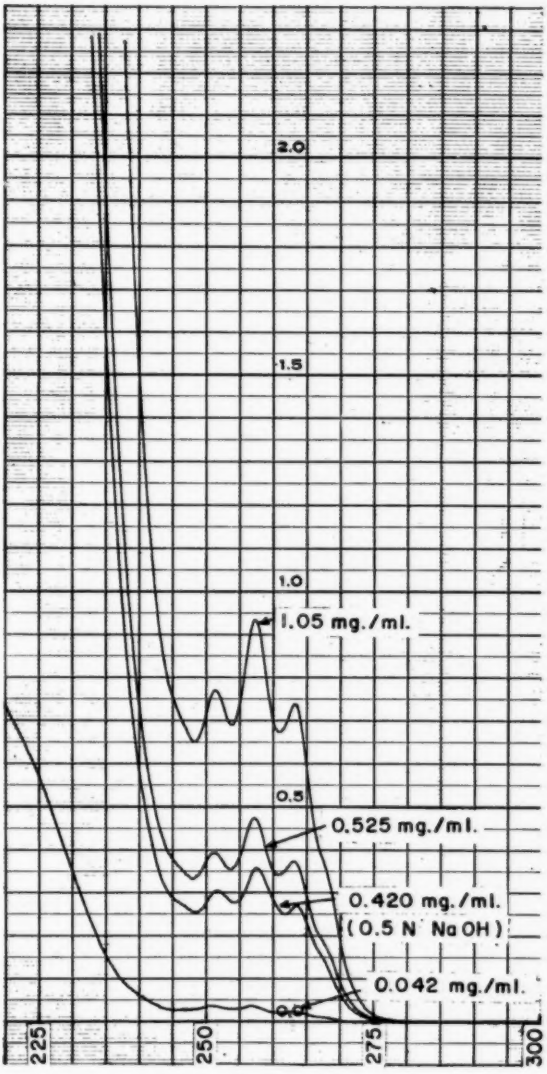

Fig. 1—Ultraviolet spectra of meperidine at four concentrations.

by an absorbancy of 0.825. If an absorbancy of 0.885 equals 1.0 mg./ml. of meperidine (or normeperidine) base, 0.825 represents a concentration of

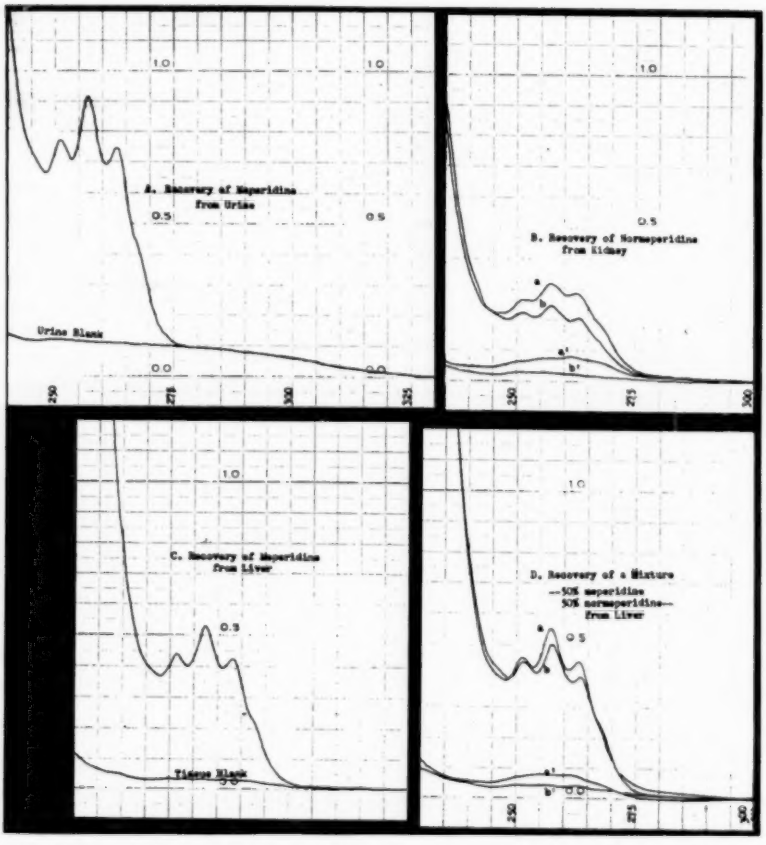

Fig. 2—A. 0.91 mg./ml. meperidine base recovered from 15 ml. urine (in 0.5 N H_2SO_4).

B. 0.25 mg./ml. normeperidine base recovered from 10 g. kidney—
a and *a'*—0.5 N H_2SO_4,
b and *b'*—made alkaline with KOH.

C. 0.56 mg./ml. meperidine base recovered from 10 g. liver (in 0.5 N H_2SO_4).

D. 0.52 mg./ml. meperidine-normeperidine mixture recovered from 10 g. liver—
a and *a'*—0.5 N H_2SO_4,
b and *b'*—made alkaline with KOH.

0.932 mg./ml. of meperidine. Since the urine contained the equivalent of 0.99 mg./ml. of meperidine base, 0.932 mg./ml. indicates a recovery of 94%. (The

actual recovery was 92%.) To compensate for recovery loss, a standard absorbancy of 0.82 should be used instead of 0.885.

Estimation of meperidine (or normeperidine) in tissue must be performed in another manner since the blank is usually not of a linear nature. The calculations should be made utilizing absorbancies of the spectra read in alkali. In general, alkali tends to decrease the blank to a point where it may be discounted entirely (see Fig. 2 B and 2 D). To illustrate this second method, the recovery of a meperidine-normeperidine mixture of liver tissue (Fig. 2, D) is calculated below. Assuming no knowledge of the actual blank, the absorbancy of curve "b" (spectra in alkali) is employed in the calculation.

$$\frac{0.50}{0.885} \times \frac{100}{75} \times 5 = 3.77 \text{ mg. total meperidine-normeperidine mixture.}$$

0.50 = absorbancy of curve "b" at 257 mμ.
0.885 = absorbancy of 1.0 mg./ml. meperidine (or normeperidine) base.
75/100 = solvent aliquot.
5 = 5 ml. 0.5 N sulfuric acid to extract solvent.

Since 3.72 mg. of the mixture (50% meperidine-50% normeperidine) were added to the liver, 3.77 mg. represents a recovery of 101%. The actual recovery—obtained by subtracting the blank at 257 mμ—was 93%.

High blanks are detected by the distortion of the phenylpiperidine spectra (see Fig. 2, B, curve "a"). If alkali does not successfully reduce these blanks, the ether re-extraction is necessary. However, it is important to keep in mind that 95% of the meperidine (or normeperidine) base is recovered from each ether extraction of an aqueous solution.

Photographs were taken of meperidine pierate crystals extracted from the "case" urine (see Fig. 3).

Discussion

Plotnikoff, Elliott, and Way (5) found that demethylation and hydrolysis were the principle detoxification mechanisms encountered in their study of meperidine metabolism. They succeeded in isolating metabolites which had the properties of normeperidine and 1–methyl–4–phenyl isonipecotic acid. Less than 10% of the total dose of meperidine was excreted unchanged in the urine over a period of twenty-four hours. Since an ultraviolet procedure based on the measurement of meperidine exclusively would be applicable only to large concentrations, a study of normeperidine was made to ascertain the possibility of increased sensitivity by inclusion both of meperidine and normeperidine in the extraction. Meperidine

and normeperidine have identical spectra, therefore, an extract which contained both parent compound and metabolite would account for a large enough fraction of the ingested dose

Fig. 3—Photomicrograph (388 x) of meperidine-picric acid (1.2%) crystals extracted from the "case" urine.

to make ultraviolet spectrophotometry feasible. Results of paper partition chromatography confirmed this assumption.

Extracts of the tissues (excluding the lung) from the case were combined after being read in the spectrophotometer, and chromatographed according to the method described by Goldbaum and Kazyak (2). An extract of lung tissue was chromatographed independently since the concentration was sufficiently high to be able to distinguish meperidine from normeperidine. Upon development overnight in a chromatocab, two distinct movements were noted with the lung tissue extract at pH 5.0 and above corresponding to the development of both meperidine and normeperidine. With the combined extract only the chromatogram at pH 6.5 showed the two different movements. The higher R_f compound resembling meperidine movement produced only a very weak spot with iodoplatinic acid which might explain the absence of any spot at pH 7.5. At the higher pH's the iodoplatinic acid reagent loses sensitivity and could conceivably have failed to detect a small concentration of meperidine. The R_f's are reproduced in Table II. With the exception of the stomach contents, the indication from chromatography is that the compound recovered from the tissues is predominantly normeperidine.

TABLE II

Chromatography of Tissue Extracts from "Case"
(By method of Goldbaum and Kazyak (2))

	R_f			
pH	3.0	5.0	6.5	7.5
Normeperidine marker (100 μg.)	0.51	0.48	0.45	0.56
Meperidine marker (100 μg.)	0.51	0.56	0.66	0.90
Extract of combined tissues from "case"	0.55	0.48	0.44	0.56
			0.62	
Extract of lung from "case"	0.51	0.45	0.42	0.55
		0.56	0.62	0.89
Extract of stomach content	0.55	0.55	0.71	0.92

Results of the present procedure were compared with those obtained from the "methyl orange procedure" of Brodie and Udenfriend (1). One gram of lung from the "case" was analyzed by the Brodie technique modified to determine

meperidine as proposed by Way et al. (6). The meperidine levels were significantly lower than those originally found by the ultraviolet procedure. Another one-gram portion of the lung was then reanalyzed by the "methyl orange procedure" with the substitution of buffer pH 8.5 (M/15 dibasic sodium phosphate) instead of the buffer pH 7.0 wash specified by E. L. Way. To check the similarity of the metabolite with the original meperidine spectra, a third specimen of lung was again analyzed by Brodie's method (Way's modification), but 0.5 N sulfuric acid, rather than methyl orange reagent, was used to extract the ethylene dichloride in the final step of the procedure. When the acid extract and buffer pH 7.0 wash were read in the spectrophotometer, the meperidine spectra was detected in both solutions. With the substitution of buffer 8.5 for buffer 7.0 in the "methyl orange procedure," sections of liver from the "case" were also subjected to analysis by both methods. The results of these comparisons are given in Table III.

TABLE III

Specimen	Meperidine concentration (expressed as mg. free base)	
	From Ultraviolet Absorption Spectra	By Methyl Orange Procedure (Brodie *et al.* (1))
Lung from "case"	0.34 mg./gm.	0.16 mg./gm.*
Lung from "case" (2nd sample)	0.23 mg./gm.* 0.12 mg./gm. (retained by buffer pH 7.0)	0.37 mg./gm.
Liver from "case"	0.17 mg./gm.	0.16 mg./gm.

From Table III, the buffer pH 7.0 wash would seem to indicate a convenient method for the separation of meperidine from normeperidine. However, the separation of these compounds, which is inferred by Table III, is not complete. With an equal volume of a buffer as low as pH 6.5, only 50% of the normeperidine was extractable from the chloroform after a ten minute shaking period. Although the buffer wash was responsible for the extraction of a considerable amount, it

* Buffer pH 7.0 wash employed as designated in the "Way modification (6)" of the "methyl orange" procedure.

does not account for all of the normeperidine metabolite extracted from the tissue.

Since normeperidine has little value as an analgesic (4), it is not likely to be encountered except as a metabolite of meperidine. Consequently, from the standpoint of toxicology, isolation of this compound is not necessary except where there is reason to doubt meperidine ingestion. Chromatography or a series of buffered extractions will effectively accomplish this separation, but unless the concentration of meperidine were high, quantitative estimation might not be possible by ultraviolet spectrophotometry. On the other hand, normeperidine, the predominant compound, should present no problem.

Interfering substances are limited to those which possess an ultraviolet absorption spectra and are not eliminated by the buffer (pH 8.5) wash or the ether re-extraction. Fresh tissue and urine specimens present no problem in this respect. Tissue from the "case" was kept frozen except for short periods during which sections were cut for analyses. In the eight months that these tissues were undergoing study prior to the publication of this data, very little change was noticed in the amount of the interfering compounds which were encountered in the initial extractions. In every instance, re-extraction with ether eliminated the contamination.

As might be expected, other compounds possess spectra similar to that of meperidine and could conceivably interfere with the analysis. Atropine, homatropine, scopolamine, amphetamine, desoxyephedrine, and ephedrine all have the characteristic maxima at 263, 257, and 251 mμ. However, the exception in the meperidine (phenylpiperidine) spectra lies in the fact that after the minima at 248 mμ, the absorbancy increases sharply below 245 mμ. This is not so with these other compounds, especially when they are made alkaline. In general, they exhibit a fourth maxima at about 246 mμ and begin their ascent (absorbancy increase) below 240 mμ. Consequently, only the very low concentrations of meperidine might offer any confusion in this respect. Again, chromatography could resolve this doubt.

In connection with interfering substances, standard solutions of meperidine can be made up from commercial prepara-

tions of Demerol. However, *m*-cresol is used as a preservative in some Demerol solutions and interferes with the meperidine spectra. To eliminate *m*-cresol completely from these preparations, the Demerol solution (pH 5.0 or lower) must be extracted with an equal volume of ether before the aqueous solution can be read in the spectrophotometer. Because meperidine salts are extremely insoluble in ether, there is no loss of Demerol.

TABLE IV

Standard Recoveries of Meperidine and Normeperidine Added to Biological Tissues and Fluids

Specimen	Quantity of Standard * Added	Quantity of Standard * Recovered	Percent of Recovery
	Meperidine		
15 ml. urine	5.4 mg.	4.9 mg.	92
10 g. liver	2.65 mg.	2.49 mg.	94
	Normeperidine		
15 ml. urine	5.5 mg.	5.1 mg.	93
10 g. kidney	0.92 mg.	0.86 mg.	93
10 g. liver	3.6 mg.	3.25 mg.	90
	50% Meperidine-50% Normeperidine Mixture		
10 g. liver	3.72 mg.	3.45 mg.	93

Summary

1. High concentrations of a combination of meperidine and normeperidine were found in the tissues and fluids of a thirty-two year old male, suspected of meperidine intoxication.

2. A rapid, simple technique for the identification and quantitative estimation of meperidine is described. This procedure is based on the ultraviolet absorption of phenylpiperidine. For the purpose of toxicology, metabolites do not interfere since those that extract under the prescribed conditions have essentially the same ultraviolet spectra as meperidine and serve to increase the sensitivity of the procedure. Chromatography provides an effective means for the separa-

* Demerol and normeperidine hydrochloride were used to prepare standard solutions, but the concentrations are expressed as mg. of free base.

tion of meperidine and normeperidine if this is necessary for metabolic and other studies.

Acknowledgment:

The author wishes to express his gratitude to the Toxicology Section of the Sixth U. S. Army Medical Laboratory for their cooperation in referring this "case" to our laboratory. Thanks are due also to Dr. M. L. Tainter of the Sterling-Winthrop Research Institute who supplied the normeperidine for this study, and to Mr. Robert Kreidler for his technical assistance.

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Book Reviews*

LABORATORY MEDICINE—HEMATOLOGY, by John B. Miale, M.D., Professor of Pathology, University of Miami School of Medicine, and Director of Clinical Pathology, Jackson Memorial Hospital, Miami, Florida. C. V. Mosby Company, St. Louis, 1958. 735 pages, 192 illustrations, 4 black and white plates, 5 color plates. $13.75.

The author has assigned himself the Herculean task of writing a three volume work on laboratory medicine for the benefit of medical students, clinicians, pathologists, and medical technologists. He aims not only to catalogue and integrate the rapidly expanding knowledge of the encompassed fields, but also to induce in the reader a logical system of thinking with reference to laboratory medicine. This is the first volume; others to follow will cover Chemical Pathology and Microbiology. He has succeeded amazingly well in achieving his goals in this monograph. The style is lucid and interesting. Difficult and controversial matters are handled in a manner which should be particularly helpful to the novice. His attitude and approach to concepts and problems are generally sound, and compatible with those of other authorities in this field. Illustrations, tables, and photomicrographs are, for the most part, pertinent, clear, and of good technical quality. Five color plates, each containing 24 photomicrographs of blood or marrow cells, are good but, unfortunately, fall short of ideal.

The first half of the book deals with general concepts of hematology, exclusive of coagulation. This is followed by a section on specific hematologic diseases with particular emphasis on laboratory aids in diagnosis, and with little or no discussion of prognosis, management, or autopsy findings.

* These book reviews have been prepared by selected authorities in the various fields of the forensic sciences, but do not necessarily represent the opinions of the editorial staff, the American Academy of Forensic Sciences, or the publisher.

The chapter on bone marrow contains a well-selected and helpful series of 37 clinical cases in which history, physical examination, laboratory findings, and discussion appear on one page facing black and white reproductions of appropriate material such as blood smear, marrow smear and marrow section, on the next. There is an excellent chapter on "Hemostasis and Blood Coagulation," a field in which the author has obviously had extensive personal experience. On the other hand, this reviewer is somewhat disappointed in the discussion of leukemia. The final 100 pages are devoted to the laboratory techniques of hematology. Details of methods preferred by the author are outlined clearly and concisely with inclusion of helpful and practical critiques.

The bibliography is given according to author under broad topic headings at the end of each chapter. Selection is good and includes many recent articles. However, significant advances of the last year or two have not uniformly been included. No attempt is made to document statements with specific bibliographic references. The index is helpful for locating general topics, but falls short with regard to cross-references and minor points.

This book is recommended as an excellent text for those who are first learning the laboratory aspects of hematology, and as a desirable addition for browsing and reference to those already familiar with the field.

William R. Best, M.D.

ACADEMY LECTURES ON LIE DETECTION, Vol. II, by V. A. Leonard, Editor, Professor of Police Science and Administration, The State College of Washington, Pullman, Washington. Charles C Thomas, Publisher, 1958. p. 156.

This book is a compilation of papers presented at the Ninth Seminar-Convention of the Academy for Scientific Interrogation at Hot Springs, Arkansas in August, 1957. The first chapter is an essay written for the Saturday Evening Post by Alva Johnston, and presents a fascinating account of Leonarde Keeler's most interesting cases. It is difficult to understand, however, why Keeler instituted a six-weeks training course for polygraph examiners when Keeler is

quoted by the author as saying that fully one year of training is necessary to qualify a competent examiner.

The remaining papers contained in this volume are particularly interesting only to persons in the polygraph field. For the most part, they are discussions of interrogation techniques in connection with the lie detector, various techniques of polygraph test administration, experiments in instrumentation and matters of special interest to the members of the Academy for Scientific Interrogation.

The editor does not purport to offer a text for the training of polygraph examiners. However, for the advanced examiner the papers present some interesting experiments and observations of men who have been in the field for several years. Unfortunately, the reader is unable to determine the extent of the case experience of each of the contributors, but several papers show considerable insight into the field of polygraph examination. For example: a paper prepared by S. R. Hathaway and C. B. Hanscom, both of the University of Minnesota, entitled "The Statistical Evaluation of Polygraph Records" is a concise statement of the problem with which all polygraph examiners are concerned. The problem simply stated is how to reduce subjective analysis of polygraph data to the point where several examiners can, on the basis of objective evidence alone, arrive at a consistent decision. The contributors suggest that the solution of this problem lies in advancing techniques of polygraph administration to the point where they are more in line with statistical methods of other fields of measurement.

It is unfortunate that some contributors made disparaging remarks about certain races and organizations. Such remarks are inapropos of scientific contributions. In spite of this, however, the volume as a whole indicates a strong desire amongst the members of the Academy to elevate the professional status of the polygraph examiner and to stimulate further acceptance of the technique as a science amongst the general public.

The reviewer would recommend its reading by persons who are in the lie-detector field.

George Wakem Lindberg, B. S., J. D.